VOLUME 38 NUMBER 5 NOVEMBER, 1954

# AMERICAN JOURNAL
## OF
# OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

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DEPARTMENTS



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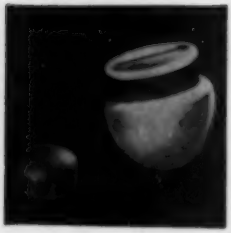

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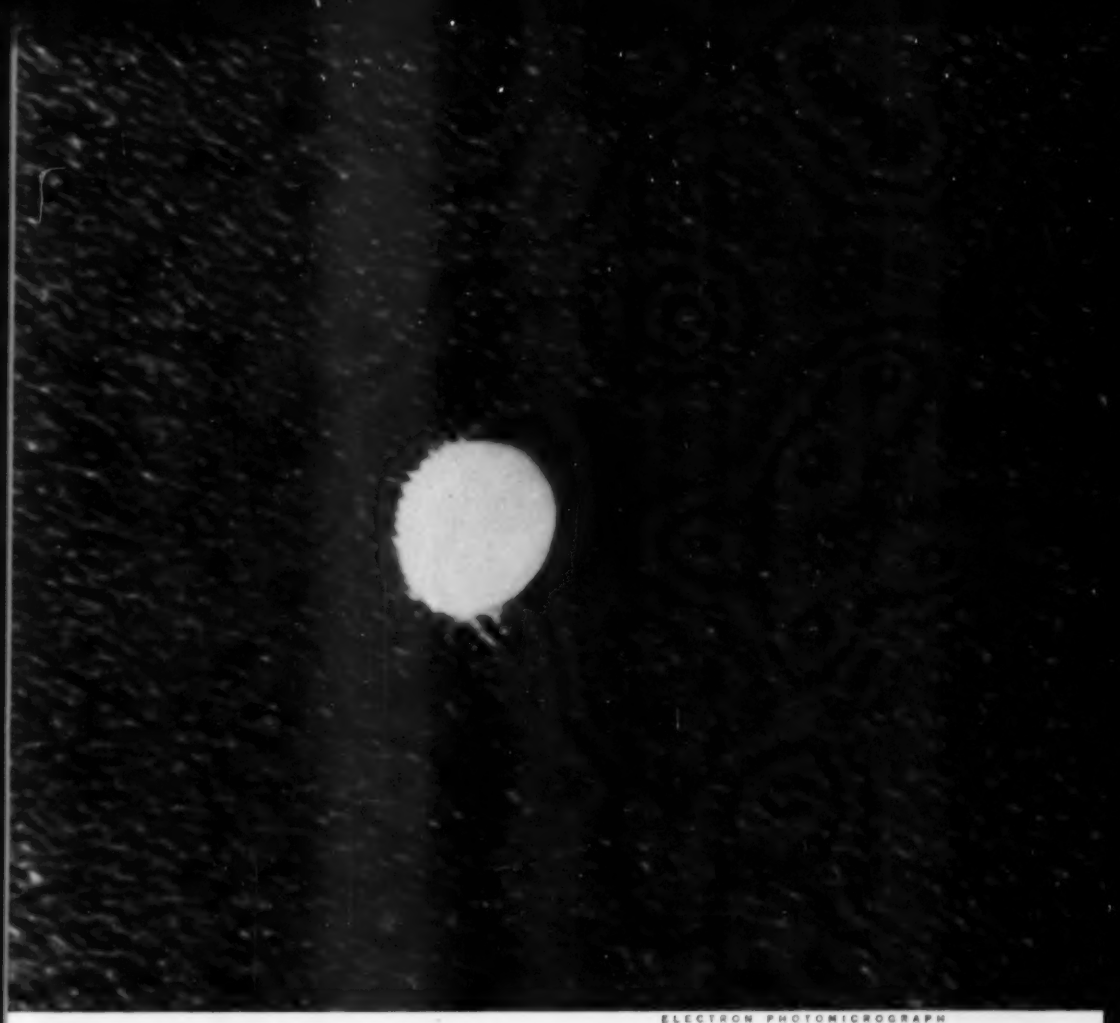

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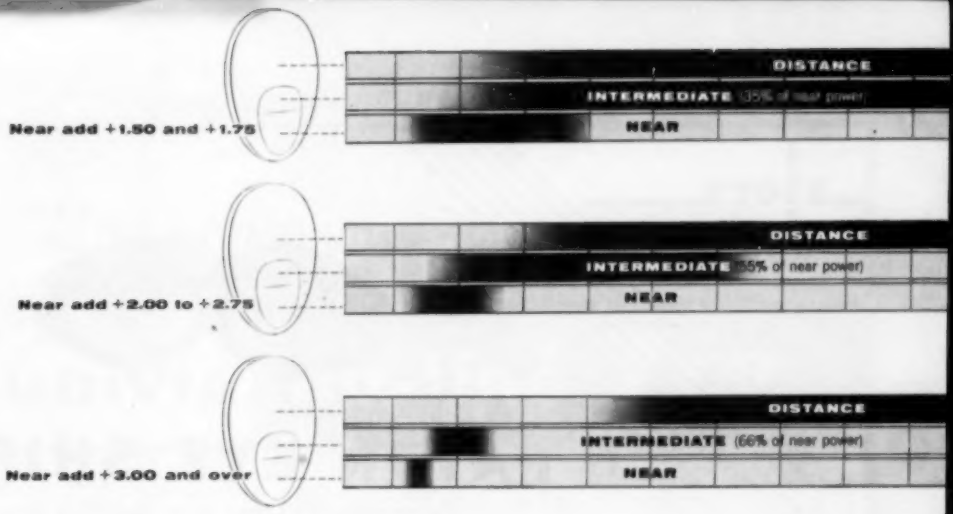


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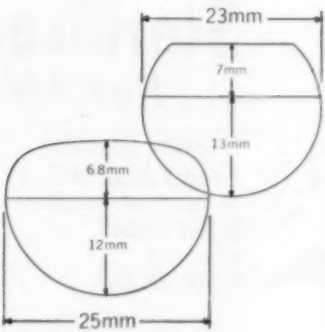


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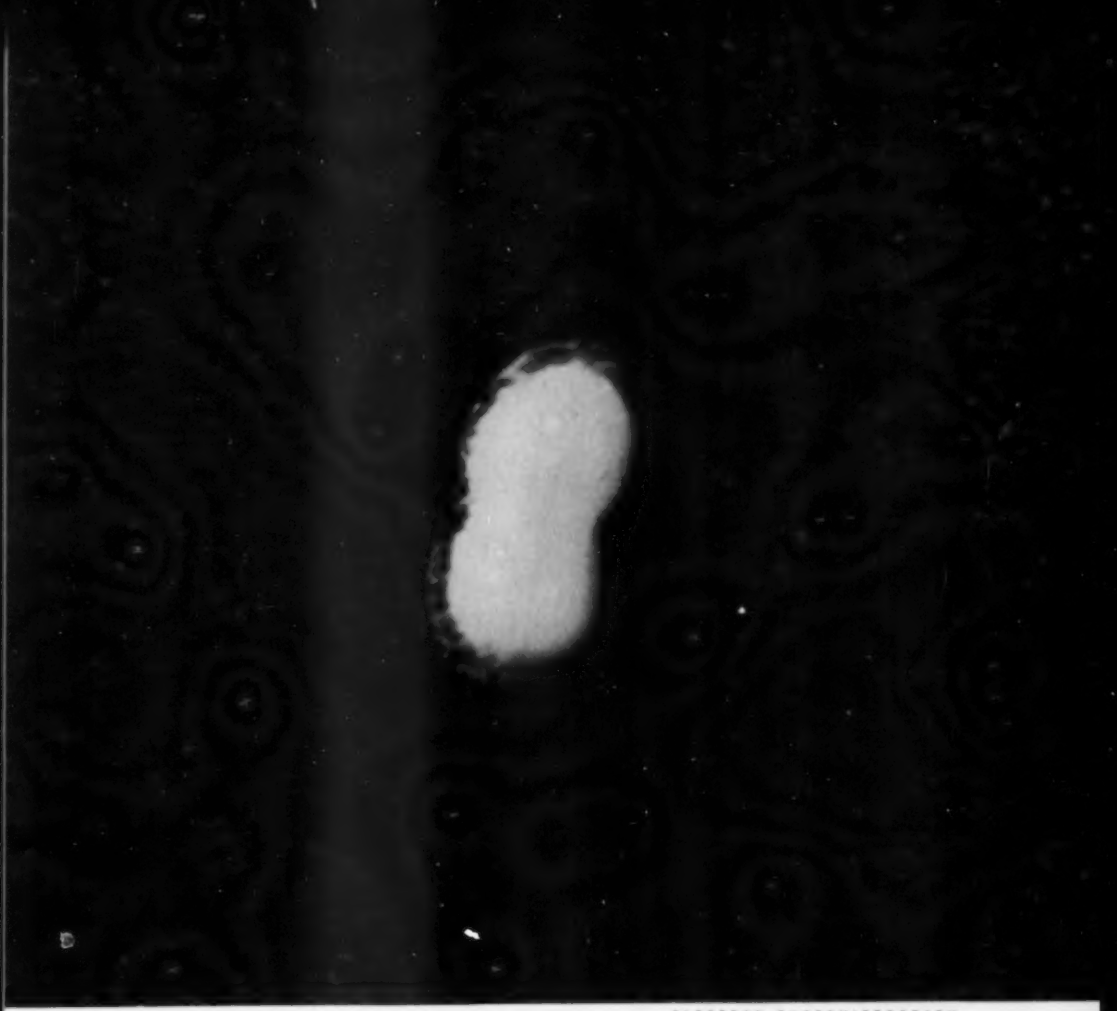

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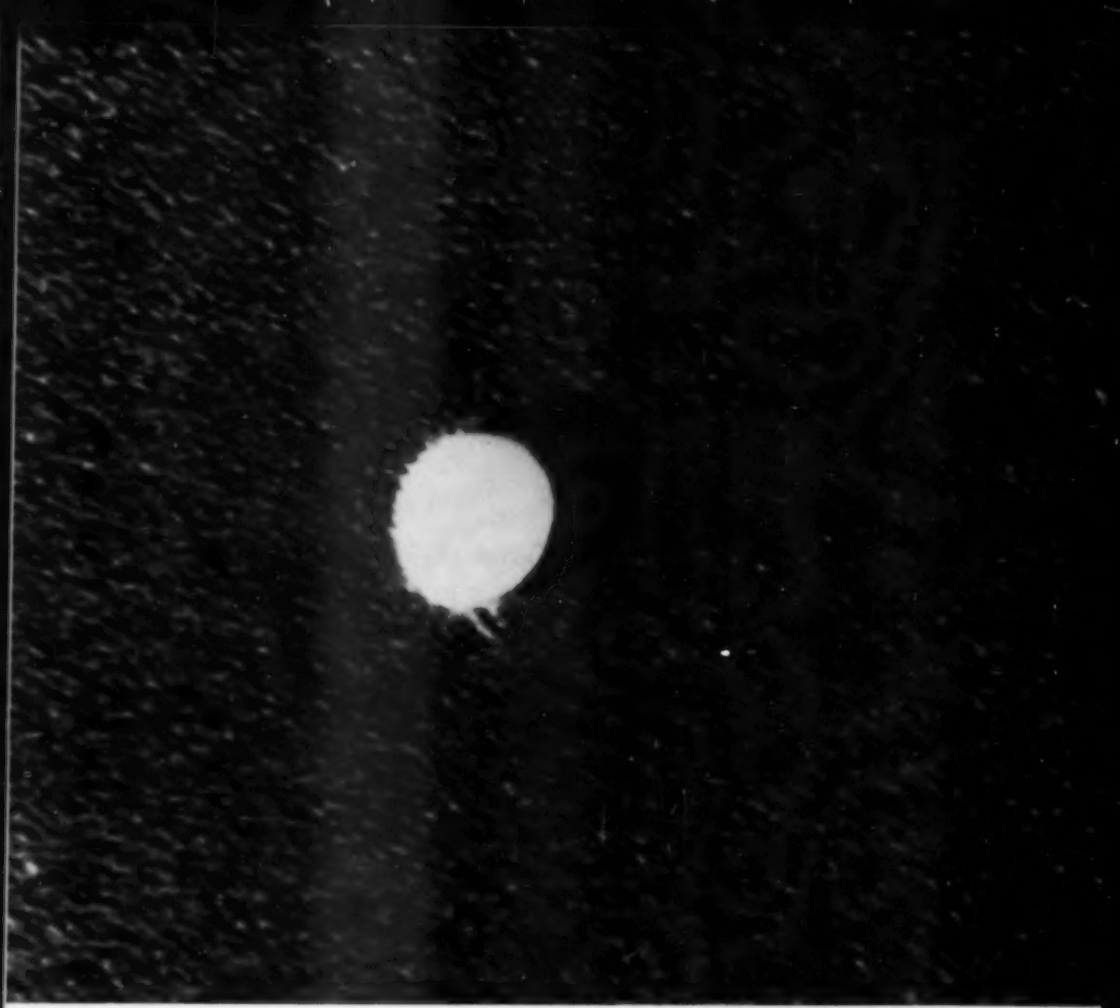

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 38 NOVEMBER, 1954 NUMBER 5

# UVEITIS WITH GLAUCOMA AND RETROBULBAR NEURITIS*

M. HARRY COHEN, M.D.
*Mount Vernon, New York*
AND
MAX CHAMLIN, M.D.
*New York*

In cases of uveitis, the occurrence of secondary glaucoma is well known, and it is commonly believed that the products of inflammation produce a mechanical blockage in the angle of the anterior chamber to cause this secondary rise in intraocular pressure. On the other hand, the association of acute retrobulbar neuritis with uveitis and secondary glaucoma is certainly less well known, though the histopathologic evidence is considerable. Nevertheless, it was pointed out in the literature, about a half century ago, that involvement of the optic nerve in its anterior or vessel-bearing portion occurs in uveitis.

In reviewing the literature, the paucity of histopathologic corroboration of such clinical findings is readily understandable, because eyes with the clinical diagnosis of optic neuritis do not ordinarily come to enucleation at the time the disease is active.

In 1911, Zeeman[1] called attention to the possibility that a ciliary-body infiltrate could be transmitted to the optic nerve through perivascular lymph channels, although we know that the existence of such lymph channels is still highly controversial.

In 1912, Straub[2] induced a papillitis experimentally by inoculating the ciliary body with tuberculosis bacilli.

In 1915, E. Fuchs[3] called attention to two avenues for the spread of infection from traumatic iritis to optic neuropathy. One was directly through the vitreous and the other by way of perivascular channels. In 1921, Meller,[4] in a descriptive article on iridocyclitis and retrobulbar neuritis, also referred to both of these possible routes of spread from the anterior uvea to the optic nerve. By careful serial sections, he traced the infiltrating cells from the ciliary body to the optic nerve, and found that they traveled through perivascular lymph channels in two eyes which had demonstrated clinically the coexistence of iridocyclitis and retrobulbar neuritis in the absence of visible ophthalmoscopic changes in the retina and choroid.

One year later, Kleinsasser[5] described five cases of iridocyclitis with several scotomas which he believed to be due to retrobulbar involvement. In the same year, Clegg[6] reported five similar cases. The only fundoscopic alteration referred to by Kleinsasser and Clegg is that of some hyperemia of the disc with relative engorgement of the veins, in one or two of the 10 cases. This disc change may well have been evidence of retrobulbar neuritis.

In 1923, Zeeman[7] again referred to anterior uveitis with optic-nerve changes. In 1931, Osterberg[8] reviewed the literature and reported a case of iridocyclitis with a five-degree central scotoma for 5/330 white, together with secondary glaucoma and a tension as high as 45 mm. Hg. This case presented no excavation of the disc but did

* From the Ophthalmological Service of the Montefiore Hospital, New York.

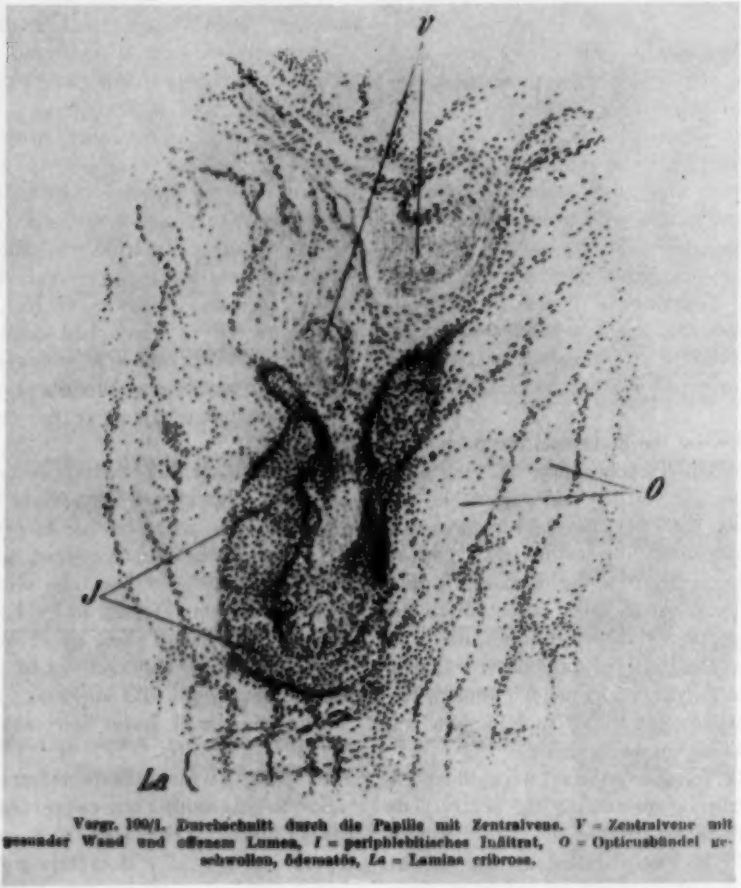
show some hyperemia. The central scotoma disappeared in six months.

In 1941, Duke-Elder[9] pointed out that inflammatory complications involving the retina and optic nerve were rare in affections of the anterior segment of the uveal tract.

More recently, Gartner[10] called attention to the frequency of optic neuritis secondary to uveitis, and pointed out that this is more marked when the lesion is near the disc.

It is to be noted from the foregoing references that we were able to find only two cases over the past 35 years in which it was possible to correlate clinical and histopathologic data (figs. 1 and 2). Most of the reported cases of uveitis in which retrobulbar neuritis and papillitis were simultaneously present were of long standing, that is, chronic cases with many of the sequelae of chronic iridocyclitis.

These showed posterior synechias, complicated cataract, seclusio and/or occlusio pupillae, vitreous opacities, keratic precipi-




Vergr. 100/1. Durchschnitt durch die Papille mit Zentralvene. *V* = Zentralvene mit gesunder Wand und offenem Lumen, *I* = peripheblitisches Infiltrat, *O* = Opticusbündel geschwollen, ödematös, *La* = Lamina cribrosa.

Fig. 1 (Cohen and Chamlin). Reproduction of a case of J. Meller,[4] showing an infiltration in the depths of the optic papilla of an eye with iridocyclitis. Meller states that, in this same eye, he found another small independent infiltrate two mm. behind the lamina cribrosa.

tates, permanent structural changes in the iris such as an ectropion uveae, sector-shaped atrophy, depigmentation, secondary glaucoma, and so forth. The diagnosis of retrobulbar neuritis was generally made on the basis of visual field changes, that is, central or paracentral scotomas in the absence of visible ophthalmoscopic changes in the fundus area corresponding to the visual field defect.

Occasionally, a case is described in the literature wherein, while the uveitis is in the early acute stage, a sudden deterioration of vision occurs with no valid explanation for such loss in the media or fundi, and such a case may well indicate neuritic involvement. A case with these findings is mentioned in the literature by Meller.[11]

All the references just cited mention retrobulbar neuritis occurring secondarily to uveitis. The reverse of this—the possibility of uveitis secondary to optic-nerve involvement—has not been mentioned, to our knowledge but it should also be borne in mind in evaluating our case.

## Case presentation

A 27-year-old white man, previously in excellent health, presented himself on January 22, 1953, with a history of "sudden onset of blurring of vision" in his left eye. This sudden onset occurred eight days prior to his examination, and there had been no improvement during that time.

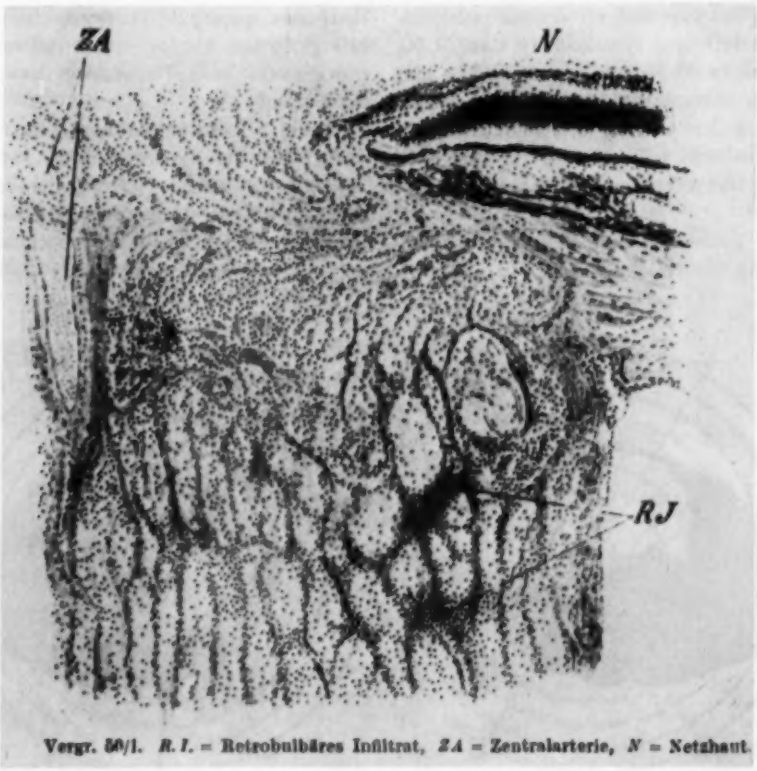



Fig. 2 (Cohen and Chamlin). Reproduction of a case of J. Meller,[4] showing retrobulbar neuritis infiltration occurring in an eye with chronic iridocyclitis.

His previous ocular history and family history were not especially contributory, although he did give a rather vague story of a slight haze before his left eye for a month or two before this sudden blurring of vision. This was quite vague. There was no history of any trauma.

Examination on the eighth day of the disease revealed that both eyes were white and quiet. The pupil of the left eye was slightly larger than the right, probably due to the mydriatics previously instilled by another eye physician.

Visual acuity in the right eye was 20/15 without correction. The right eye showed no evidence of any aqueous abnormalities and examinaton of the fundus showed that the disc had a rather hazy border nasally, with a good cup and no venous pulsation.

In the left eye, visual acuity was 20/60, correctible to 20/50 with a pinhole. The tension was elevated to fingers. The fundus revealed a disc similar to that of the right eye, again with no venous pulsation and a cup that was even smaller than that of the right eye.

There seemed to be a yellowish haze on the papilla which extended toward the fixation area as well. This hazy appearance of the disc could not be interpreted as being definitely abnormal, nor could it be said that it looked very different from the other eye.

Slitlamp examination of the left eye revealed a mild to moderate flare, and several floaters. There were no keratic precipitates, and under maximal dilatation with 10-percent neosynephrine hydrochloride, no evidence of posterior synechias was found. A prolonged examination of the entire fundus revealed no evidence of any choroidal or retinal inflammation.

The intraocular pressure (Schiøtz) measured 40 mm. Hg in the left eye and 22.5 mm. Hg in the right eye. There was no ciliary tenderness and no photophobia. However, there was some pain on retropulsion of the left globe into the orbit, but not on ocular movements. Transillumination was normal throughout.

Visual field examination revealed a large nerve-fiber bundle defect which was quite dense, and extended from the blindspot downward, both temporally and nasally, and breaking through to the very periphery (fig. 3a). Nasally, there was a very strong sug-

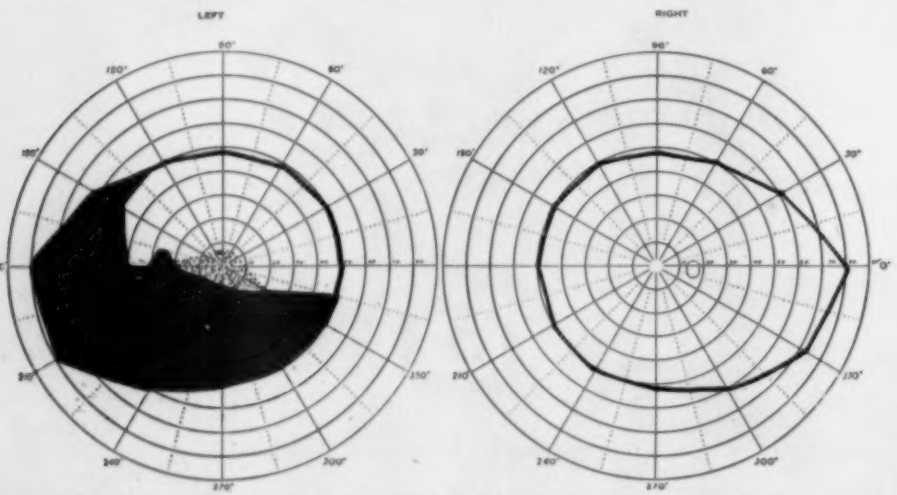



Fig. 3A (Cohen and Chamlin). Visual fields in the present case on the eighth day of illness. Visual acuity was: O.D., 20/15—1; O.S., 20/60. Peripheral field for 2/330 white, with relative encroachment on fixation.

gestion of a sharp horizontal line, indicating the end of a lower nerve-fiber bundle defect. This nerve-fiber bundle defect skirted fixation, but there was a less dense area of involvement of the fixation area, accounting for the loss of central visual acuity.

Re-examination of the intraocular pressure after maximal dilatation with a 10-percent neosynephrine, revealed a slight fall in pressure. Homatropine hydrobromide was then instilled, and 24 hours later the tension was 19 mm. Hg in the right eye and 15 mm. Hg in the left eye. This response to homatropine was considered as some corroboration that the glaucoma was secondary, rather than of a primary type. Atropine sulfate was then instilled, and prescribed for use at home.

Five days later, visual acuity was 20/25 in the affected eye, and the aqueous no longer showed any flare. There was a corresponding improvement in the visual field at fixation, and the tension was 19 mm. Hg.

Approximately five weeks after the onset of this disease, a venous pulsation appeared on the disc of the left eye and its appearance suggested the possibility that there had been retrobulbar inflammation with subsidence and the return of venous pulsation. The vision at that time had improved to 20/20, and the tension was within normal limits, with only an occasional rise. While the nerve-fiber bundle defect below fixation remained, central involvement had disappeared completely (fig. 3b).

During the succeeding 10 months, atropine was used intermittently as the flare and tension reappeared from time to time. The tension never went any higher than 29 mm. Hg during these months. On most occasions, the appearance of the flare was not accompanied by floaters in the anterior chamber, regardless of the state of the tension.

At the present time, one year after the onset of this illness, a fairly large and dense residual nerve-fiber bundle defect remains, practically the same as it was on the 13th day after the onset. This defect skirts fixation but does not reach it, and breaks through to the lower periphery.

Visual acuity fluctuates between 20/20 and 20/25, and intraocular pressure varies from 19 to 25 mm. Hg in the left eye. In the right eye it measures 19 mm. Hg. The left eye has remained white and quiet, and the patient has

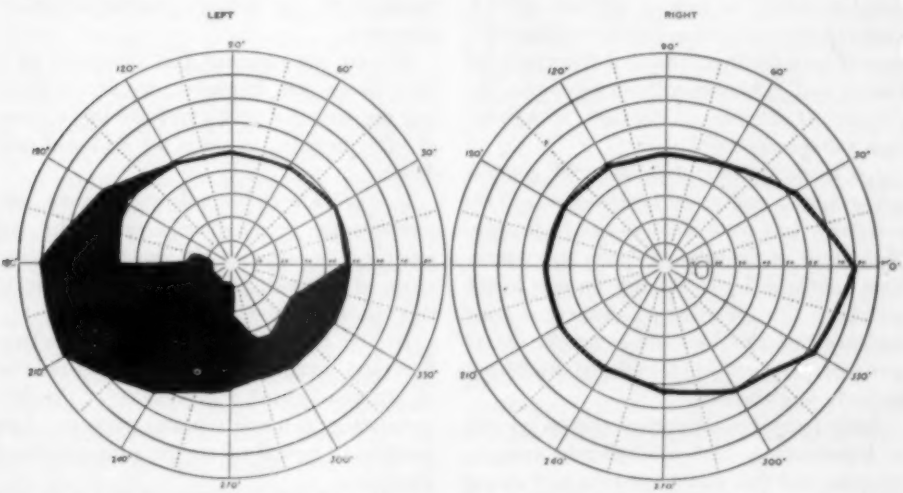



Fig. 3B (Cohen and Chamlin). Five weeks after onset. Visual acuity was: O.D., 20/15; O.S., 20/20. Fields for 2/330 white show clearing of central area. Most of this clearing was seen by the 13th day of illness.

had no complaints, except for the fact that he is aware of the visual defect in the lower half of the field of vision of the left eye.

## Comments

In evaluating the above case, several diagnostic possibilities present themselves. These will be listed in the order of their likelihood.

1. Uveitis with secondary glaucoma, and complicating retrobulbar neuritis.
2. Retrobulbar neuritis with complicating uveitis, and glaucoma related to the uveitis.
3. Primary glaucoma with coincidental uveitis and retrobulbar neuritis.
4. Glaucomatocyclitic crises with coincidental optic neuritis.

The diagnostic possibilities will be discussed in the reverse order of what we consider their likelihood.

4. *Glaucomatocyclitic crises.* Several factors make one consider this possibility. Essentially, these are the lability and unilaterality of the increased intraocular pressure, as well as the inconsistency of objective and subjective findings. On the other hand, the fact that there was a loss of visual field and that there were no actual "crises," with a complete return to normal between attacks, would be very much against such a diagnosis, according to the description of this entity by Posner and Schlossman.[18] Furthermore, the absence of keratic precipitates is against such a diagnosis.

3. *Chronic simple glaucoma with complicating uveitis and retrobulbar neuritis.* This possibility was considered especially because of the type of field defect; namely, a nerve-fiber bundle defect breaking through to the periphery. On the other hand, there is good evidence that this nerve-fiber bundle defect occurred very suddenly, and this would not be likely in glaucoma.

Aside from this, the patient responded well to homatropine, and subsequently even to atropine, and this would also be a very strong point against primary glaucoma. Furthermore, the essential pathologic process of glaucoma would not readily account for a central scotoma. Finally, even if there were some sort of flare with glaucoma, it would certainly not be likely to occur with a tension as low as 19 to 25 mm. Hg.

2. *Retrobulbar neuritis with complicating uveitis and glaucoma.* This was another possibility that occurred to us, inasmuch as there was a sudden onset of marked blurring of vision, with a nerve-fiber bundle defect compatible with an attack of retrobulbar neuritis.

While we are not aware of any reports of uveitis occurring secondarily to retrobulbar neuritis, it may be considered as a possibility. Perhaps the finding of a faint flare and a few cells in the aqueous may occur with a retrobulbar neuritis but is not often looked for.

On the other hand, if such were the case here, we could well expect that, with the subsidence of the retrobulbar neuritis, certainly the mild flare in the aqueous would have disappeared completely. Instead, it continued to recur, as is typical in low-grade uveitis. Furthermore, the mild type of flare that would occur from such a complication would hardly be likely to produce secondary glaucoma.

We do not consider this diagnosis as a very likely one. However, we are examining the aqueous very carefully in all cases of optic neuritis, to see if we find such evidence.

1. *Uveitis with secondary glaucoma, and complicating retrobulbar neuritis.* In our opinion, this appears the most likely diagnosis in this case. Several factors point to this diagnosis as a good possibility.

In the first place, there was a definite flare and some floaters, and the instillation of atropine caused a subsidence of the flare and a drop in the intraocular pressure. This reaction is typical of uveitis with secondary glaucoma.

The fact that the flare recurred from time

to time, with fluctuation in tension, is again very typical of uveitis. Furthermore, the patient gave a story of a very faint haze before that eye for a month or so prior to the sudden blurring of vision caused by the dense field defect below. This could very well have been due to a low-grade uveitis, with the resultant slight haze prior to the onset of retrobulbar involvement.

Actually, the clinical picture was characteristic of retrobulbar neuritis in several ways. Thus, the onset of loss of vision was sudden, and while the scotoma was not a caecocentral one, it was a nerve-fiber bundle defect involving fixation. Such types of visual-field defects have been shown to occur not too infrequently in optic neuritis, in a recent paper by one of us.[13] The tenderness on retropulsion of the globe was another point for retrobulbar inflammation, in this case. Aside from the mode of onset, the subsidence of the retrobulbar involvement, as evidenced by the clearing of the central defect on the ninth day, together with persistence of the more dense peripheral defect, is again quite typical of retrobulbar neuritis.

There are several factors which are against a diagnosis of uveitis. One of these is the fact that the intraocular pressure would rise to 25 or 28 mm. Hg at times, even when the aqueous appeared relatively clear. Furthermore, there were never any posterior synechias and no ciliary tenderness or photophobia.

On the other hand, the uveitis was of a mild nature, though sufficiently severe to produce secondary glaucoma and retrobulbar involvement in this particular patient.

It would be interesting at this point, to note that in 1934, Selinger,[14] while working on some features of the differential diagnosis between papillitis and papilledema, observed that, in papillitis, the aqueous humor contained an elevated concentration of protein. This observation may throw some light on our present concepts of the association of optic neuritis with anterior-segment disease. Though this reference is the only one of its kind that we found, it brings to light the possibility already mentioned, that is, of an aqueous inflammatory reaction being caused by an optic neuritis, or perhaps even retrobulbar neuritis.

It would be difficult for the clinician to say how frequently some aqueous changes occur secondary to retrobulbar neuritis, since it is quite likely that, when a case of retrobulbar neuritis is seen, the examiner is more apt to spend his time studying the disc ophthalmoscopically and examining the visual fields, rather than studying the aqueous. Therefore, slitlamp examination is urged in all cases of optic neuritis and retrobulbar neuritis, for the possible discovery of secondary uveal reaction.

Although we considered the possibility of uveal reaction secondary to retrobulbar neuritis, we do feel that the most likely diagnosis in this case is that of uveitis with secondary glaucoma and complicating retrobulbar neuritis. While the literature shows few reports of this entity, it is likely that it occurs more frequently than is generally realized.

It may well be that in cases of uvetitis where there is a good deal of loss of visual acuity this is not due to the hazy media alone but may be, at least in part, due to central or caecocentral scotomas, and especially paracentral scotomas, as a result of the secondary optic neuritis or retrobulbar neuritis.

## Conclusions

A case is presented which showed evidence of anterior uveitis, glaucoma, and retrobulbar neuritis.

The clinical picture is most compatible with primary uveitis and a secondary glaucoma, as well as retrobulbar neuritis secondary to the uveitis. It is therefore urged that, in cases of uveitis, the patients be watched very carefully for loss of visual field, as an indication of optic-nerve involvement.

*117 South 2nd Avenue.*
*8 East 77th Street (21).*

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# A FURTHER STUDY OF THE ANTERIOR ANGLE

## In anterior-segment inflammation of the eye


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In a previous paper,[1] the gonioscopic findings of the angle of the anterior chamber in several conditions of inflammation involving the anterior segment of the eye were described.

The study was undertaken not only to see what visible effects on the angle such inflammations would have but also to try to evaluate such changes in terms of function, especially on the future possibility of glaucoma.

These several cases reported are a follow-up of cases described in the previous paper. The numbers in parentheses represent the case numbers of the same patient described in the previous report.

### Case 1 (1) (fig. 1)

A. H., a white man, aged 19 years, came to the Brooklyn Eye and Ear Hospital, service of Dr. W. V. Moore, on September 19, 1950, because of blurred vision, slight pain, and redness of the left eye for about a week.

*Examination.* The right eye was normal and vision was 20/20. The left eye had a moderate circumcorneal injection, cloudy cornea, and a deep anterior chamber. The tension was: R.E., 20 mm. Hg (Schiøtz); L.E., 65 mm. Hg (Schiøtz). With the slitlamp, many keratic precipitates were found on the posterior corneal surface. Laboratory examinations, including a Mantoux test, were negative.

He was placed on cortisone and atropine drops for a few days but without improvement. The cortisone was, therefore, discontinued and foreign-protein therapy was instituted. As the eye did not respond to this treatment, he was put on tuberculin therapy.

The eye reacted favorably and in about four months, the eye became white, the tension normal, but the keratic precipitates and the aqueous flare persisted for many months later.

*The first gonioscopic examination* on October 17, 1950, showed the angle in the right eye to be widely open so that the ciliary zone was visible. There was a light pigment on the posterior trabeculum on the nasal side only. The left angle was seen through a haze and appeared to be fairly wide and with a considerable amount of pigment on the posterior trabecular zone in the lower angle, less on the nasal side and apparently none in the upper or temporal angle.

On March 14, 1951, gonioscopy of the left eye revealed a shallow angle below but moderately wide elsewhere. There was a considerable amount of pigment deposited on the trabecular zone. Above, from the 2- to 3-o'clock positions, there was a fine white veil-like tissue which extended from the root of the iris to the anterior trabeculum. The details

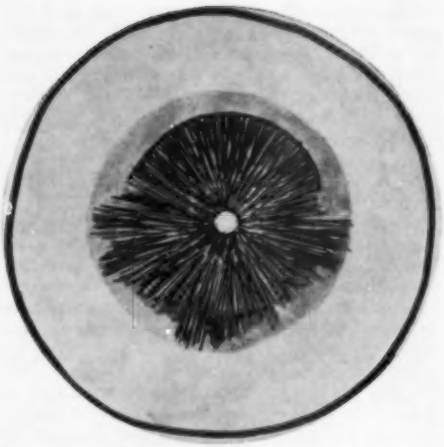

Fig. 1 (Kravitz). Appearance of left eye in Case 1.

of the structures beneath this tissue could be faintly made out.

The left eye has remained white and the intraocular pressure within normal limits. At the last gonioscopic examination, August 16, 1953, there was a decided change in the appearance of the angle:

Above, the angle was fairly wide and free of pigment excepting that the region of the canal of Schlemm was outlined by a light reddish brown pigment. Below, the angle was very shallow with dense blackish brown pigment on the small part of the anterior trabecular zone still visible. There were many fine capillaries visible on the extreme periphery of the iris. Most of these were horizontally placed, some were radial and dipped into the recess of the angle.

Laterally, the angles were moderately shallow, with dark-brown pigment deposited on the anterior and posterior trabeculum. The veil-like tissue previously described was not present but, interspersed among the pigment on the trabeculum, were many fine white dustlike precipitates. There were many fine peripheral adhesions in both lateral angles and below.

CASE 2 (5)

M. G., a white woman, aged 58 years, came to the office 10 years ago, with a history of occasional pain in the right eye for many years. At times, she saw "rainbows" before both eyes.

*Examination* revealed vision to be 20/20 in both eyes but there was deep cupping of both discs and considerably constricted fields. The intraocular pressure was: R.E., 55 mm. Hg (Schiøtz); L.E., 45 mm. Hg. As miotics did not lower the intraocular pressure, a trephining operation was performed on both eyes at the Brooklyn Eye and Ear Hospital on September 14, 1943. After operation, the intraocular pressure became stabilized around 20 mm. Hg (Schiøtz) and the vision was 20/25, corrected, in both eyes.

On February 7, 1949, she came to the office with an infection around the trephine opening of the right eye. The cornea was hazy, the conjunctiva chemotic, and there was some hypopyon in the anterior chamber.

She was admitted into the Brooklyn Eye and Ear Hospital where she was treated with a combination of fever therapy and large doses of sulfadiazine and was discharged as cured on the 10th day. Vision in that eye was reduced to 20/70 and the intraocular pressure was normal.

A few days later, the intraocular pressure went up to 45 mm. Hg (Schiøtz) and, although the trephine opening seemed to be closed by a dense scar, a one-percent solution of Carcholin brought the intraocular pressure down to 25 mm. Hg. This pressure was maintained on Carcholin and always went up to 35 mm. Hg and even to 45 mm. Hg if she neglected, for one reason or another, to put drops into the eye.

*Gonioscopic examination* revealed a veil-like exudate in the upper angle which extended from the anterior lens surface to the anterior trabecular zone. Although this organized exudate did not seem dense, the structures behind it, such as the trephine opening, could not be seen. The rest of the angle was very narrow.

After the examination in 1950, there was progressive loss of vision to light perception and the base pressure remained consistently above 35 mm. Hg (Schiøtz). In spite of this increase in pressure, the eye has remained white and painless and there has been no change in the gonioscopic appearance of the angle.

CASE 3 (11) (fig. 2)

S. F., a white woman, aged 65 years, came to the office on September 17, 1949, with a history of recurrent iritis in both eyes for which she had been under treatment for over nine months.

*Examination* disclosed many keratic deposits on the posterior surfaces of both corneas, a moderate aqueous flare, and a few posterior synechias. She was discharged as cured in January, 1950. During treatment, the intraocular pressure was never above 15 mm. Hg (Schiøtz).

A few weeks later, she had a recurrence of the iritis in both eyes.

*Gonioscopic examinations* on February 6, 1950, revealed a narrow angle in the right eye but no peripheral adhesions and no pigment on the trabeculum. The left eye had a moderately wide angle above with a small amount of pigment on the trabeculum and pigment outlining the canal of Schlemm. The lower angle was very shallow with dense pigment deposits on the small portion of the trabeculum still visible and there was a fine white flocculent precipitate on the pigment. The lateral angles were moderately shallow with a considerable amount of pigment on the posterior trabeculum.

Since the last report, she has had two attacks of

Fig. 2 (Kravitz). Appearance of left eye in Case 3.

iritis in both eyes. One, in May, 1952, and the second somewhat more severe in December, 1952.

The last gonioscopic examination, April 3, 1953, showed a slight progression of the pathologic process in the angles. More pigment was deposited on the trabecular zones and the angles were shallower. In spite of this, the intraocular pressure has never been above 15 mm. Hg (Schiøtz).

CASE 4 (fig. 3)

R. S., a woman, aged 33 years, came to the office because the right eye had been red and painful for three days. She had had a similar but milder attack four weeks before which subsided without treatment after five days.

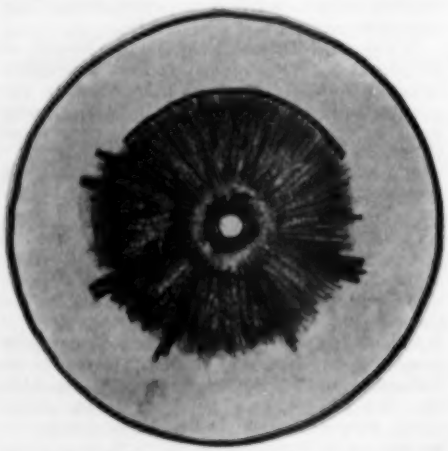

Fig. 3 (Kravitz). Appearance of right eye in Case 4.

*Examination.* The right eye was moderately congested and there was a two-plus flare in the aqueous but no keratic precipitates on the posterior cornea. When the pupil was fully dilated, many fine posterior synechias became visible. These ruptured easily with maximum dilatation of the pupil. She was discharged as cured on June 5, 1948.

On January 3, 1953, she had a recurrence of the iritis in the right eye and again without keratic precipitates.

*Gonioscopy.* Right eye: There was a very wide angle above so that the ciliary zone was easily visible. Laterally and below, the angles were moderately shallow without any pigment on the trabecular zone but there were a few scattered fine peripheral adhesions. The angles in the left eye were entirely normal.

She was discharged as cured on March 7, 1953. On July 1, 1953, she again had a recurrence of the iritis in the right eye. Gonioscopy however was not done, as the iritis subsided in a few days and she did not return.

On July 21, 1953, she returned with a mild iritis in the left eye for which she was discharged as cured on August 17, 1953.

*Gonioscopy* on the latter date showed a marked progression of the abnormal findings in the right eye as compared with the previous examinations.

The lower angle had become quite shallow and there were dense pigment deposits on the small part of the anterior trabecular zone still visible. Laterally the angles were shallow and there was light pigment on the posterior trabecular zone, with scattered light pigment on the anterior trabecular zone. Above, the angle was wide and pigment outlined the canal of Schlemm.

The left eye was entirely normal. The intraocular pressure in both eyes was 22 mm. Hg (Schiøtz) at all times.

CASE 5

L. L., a man, aged 62 years, came to the office on June 6, 1953, with a history of blurred vision and severe pain in the right eye for two days.

The right cornea was edematous, there were many folds in Descemet's membrane, many striate infiltrates in the corneal stroma, and a few keratic precipitates on the posterior cornea. The pupil was widely dilated and fixed though no medication had been instilled. The fundus could not be visualized. The intraocular pressure was: R.E., 9.0 mm. Hg (Schiøtz); L.E., 12 mm. Hg. He was able to distinguish shadows.

With therapy, the eye cleared rapidly so that vision improved to 20/20 and the intraocular pressure rose to 20 mm. Hg (Schiøtz). The pupil, however, remained widely dilated and fixed so that on July 20, 1953, pilocarpine (two percent) to be instilled nightly was prescribed. The pupil contracted slowly so that, at his most recent visit, August 22, 1953, it was smaller than that of the left eye. The iris on each side of the pupil laterally was markedly atrophic and the intraocular pressure

varied between 18 and 20 mm. Hg (Schiøtz).

*Gonioscopic examination* on August 22, 1953, showed:

Right eye: The upper angle was wide though no ciliary zone was visible. The nasal and temporal angles were moderately shallow with light pigment deposits on the posterior trabeculum. Below, the angles were very shallow with dense pigment deposits on the visible anterior trabeculum and a few peripheral adhesions.

Left eye: Very wide angles above, moderately shallow angles laterally and below. There were no pigment deposits on the trabeculum.

CASE 6 (figs. 4 and 5)

J. K., a man, aged 72 years, came to the office on May 19, 1953, with a history that, a week ago, he was standing near a cooler that was circulating cool air. The following day, the right eye began to pain and was followed in a few hours by blurring of vision. The blurred vision became progressively worse and the pain constant, moderate during the day and severe at night.

*Examination.* The right eye was considerably congested and there was superficial vascularization of the cornea around the entire circumference for between two and three mm. from the limbus. The lower half of the anterior chamber was full of blood and the upper half full of a gelatinous exudate.

Under therapy, the eye showed continuous improvement but no gonioscopic examination was possible until June 2, 1953.

*Gonioscopic examination* showed:

Right eye: The upper angle was wide but no ciliary zone was visible. There was a light pigment deposit on the posterior trabeculum. Nasally the angle, likewise, was moderately wide but the pigment on the posterior trabeculum was heavier.

Fig. 4 (Kravitz). Appearance of right eye in Case 6.

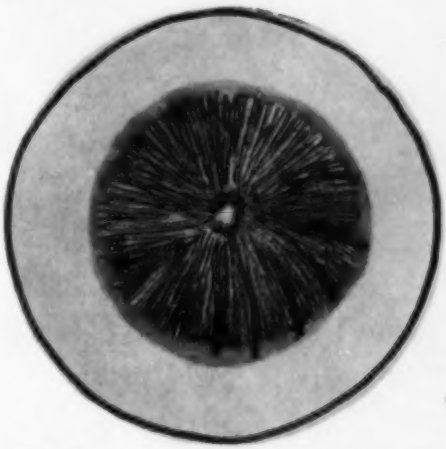

Fig. 5 (Kravitz). Appearance of left eye in Case 6.

Temporally the angle could not be well visualized because of a veil which made the details very hazy. Below, the angle was moderately shallow with considerable and dense pigment on the posterior and anterior trabeculum.

Left eye: Showed exactly the same findings as the right except for the absence of the veil on the temporal side. This eye, which was not inflamed, showed a few peripheral synechias temporally and below.

The intraocular pressure in botn eyes was 18 mm. Hg (Schiøtz). The gonioscopic picture has not varied from the description just given.

CASE 7 (figs. 6 and 7)

G. G., a woman, aged 42 years, came to the office on December 13, 1951, with a history of having been treated for an iridocyclitis of the right eye for over six months.

*Examination.* Vision was: R.E., 20/25, with correction; L.E., 20/20, with correction. The right eye had a moderate circumcorneal injection, the cornea was slightly edematous with many fine keratic deposits on the posterior cornea and an exudate on the anterior lens surface in the pupillary area. The left eye was entirely normal. The intraocular pressure in both eyes was 20 mm. Hg (Schiøtz).

*Gonioscopic examination* showed:

Right eye: The upper angle was wide though no ciliary zone was visible. There was a light pigment deposit on the posterior trabeculum and there was blood outlining the canal of Schlemm. Nasally, the angle was shallower. There was a light pigment deposit on the anterior trabeculum and a heavier deposit on the small portion of the posterior trabeculum visible. The temporal angle was slightly wider than the nasal and pigment on the trabeculum was not quite as dense. Below, the angle was ex-

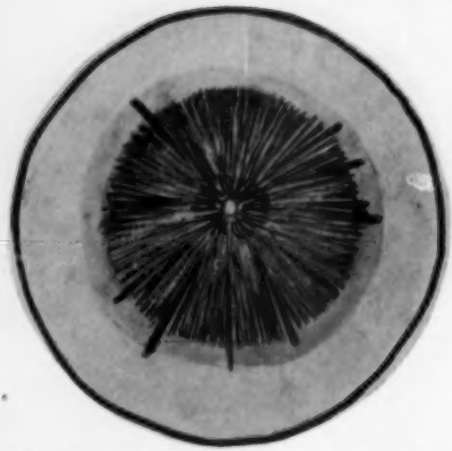

Fig. 6 (Kravitz). Appearance of right eye in Case 7.

tremely shallow so that only a small portion of the anterior trabeculum was visible, and this was heavily pigmented.

Left eye: The angles were wide all around and without pigment deposits on the trabeculum.

She was placed on cortisone drops and given a course of fever therapy and seemed to be progressing favorably. On October 15, 1952, she returned because of severe pain in the right eye. The eye was more congested and the keratic precipitates had increased both in number and size. There was also some vascularization of the iris.

*Gonioscopic examination* showed a marked progression of the condition previously described. The upper angles were still wide but the pigment on the trabeculum was darker and heavier in apperance. Laterally, the angles were shallower, the pigment on the trabeculum denser and there were a number of peripheral adhesions, more on the nasal side. Below, the angle was practically absent. In spite of this, the intraocular pressure was still 20 mm. Hg (Schiøtz).

On May 25, 1953, the lens began to show posterior cortical and capsular changes. Gonioscopic examination showed little change over that of the previous examination. Possibly, the pigmentation in the angles was heavier. The intraocular pressure did not vary from that reported at the previous examination.

Since all other therapy had failed, tuberculin therapy was advised but, because of the patient's reluctance, was not started until July 22, 1953.

The keratic precipitates have become less in number, the eye whiter and there have been no episodes of pain since starting the tuberculin therapy.

CASE 8 (fig. 8)

M. F., a man, aged 57 years, came to the office on March 21, 1953, with a history of blurred vision with the left eye for over a month and blurred vision with the right eye for several days. At no time did either eye give him pain.

*Examination.* Right eye: There was a slight edema of the cornea and a moderate number of keratic deposits on the posterior cornea. The pupil dilated irregularly.

Left eye: The corneal edema was more marked and there were many keratic precipitates on the posterior cornea. The pupil dilated very irregularly and with difficulty. The intraocular pressure was 18 mm. Hg (Schiøtz) in both eyes.

*Gonioscopic examination* showed:

Right eye: The angles were wide all around. The

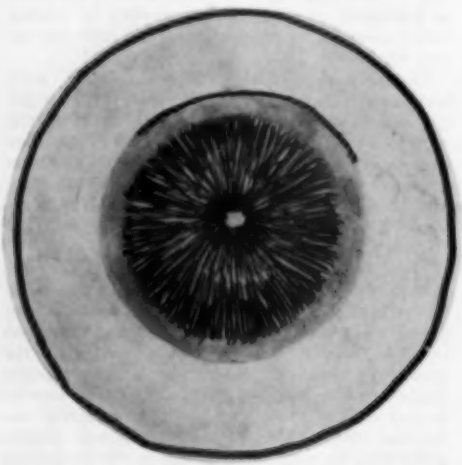

Fig. 7 (Kravitz). Appearance of left eye in Case 7.

Fig. 8 (Kravitz). Appearance of left eye in Case 8.

upper angle was entirely free of pigment. Elsewhere there was a good deal of pigment on the posterior trabeculum.

Left eye: Could not be visualized until May 19, 1953; at that time, the upper angle was found to be wide, there was blood in Schlemm's canal and a small amount of pigment on the posterior trabecular zone. A few fine peripheral adhesions were present. Laterally, the angles were shallower and only the anterior portion of the posterior trabeculum could be visualized. A considerable amount of pigment was deposited on the posterior trabeculum and less on the anterior trabeculum. Below, the angle was very shallow so that only the anterior trabecular zone could be seen. This had a dense deposit of pigment on it.

He was discharged as cured on September 15, 1953.

*Gonioscopic examination* showed:

Right eye: The angles were still wide. The pigment on the posterior trabeculum was denser and there was some pigment on the anterior trabeculum in the upper angle. Fine peripheral adhesions were present all around, most numerous below, less numerous laterally and very few above.

Left eye: With the exception of a heavy pigment deposit on the trabeculum, there were no changes in the angle.

The intraocular pressure on all examinations remained unchanged.

CASE 9 (15)

M. E., a woman, aged 55 years, had been under my care since May, 1948, for recurrent attacks of iritis and scleritis in the right eye. Some of these attacks were very protracted and severe. She was found to be allergic to many allergins.

*The gonioscopic examination* in the previous report was:

The upper angle was obliterated by a white exudate so that no details were visible. The angle on the temporal side was open but was encroached upon by the cystic iris. Nasally, the angle was moderately shallow and below, wide open. No pigment deposits were present on the trabeculum.

Since then, she had had three attacks of iritis, all mild, the last on November 21, 1952. There had been no progression of the pathologic process in the angle and no increase in intraocular pressure.

CASE 10 (9) (fig. 9)

C. D., a young man, aged 18 years, came to the office on December 20, 1945, with a history of having been struck by a flying object in the left eye, the day before.

*Examination* disclosed a ruptured cornea, traumatic cataract, and an abscess of the cornea. He was admitted into the Brooklyn Eye and Ear Hospital and, with appropriate therapy, the infection cleared. A month later, a linear extraction was performed and though there was a secondary membrane present, vision in that eye was 20/20 corrected. The membrane continued to become

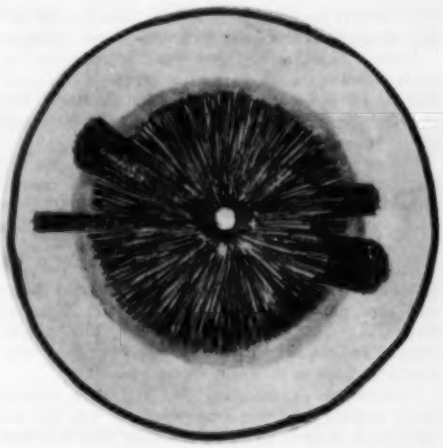

Fig. 9 (Kravitz). Appearance of left eye in Case 10.

denser with a corresponding loss of vision to 20/70. On February 23, 1947, a discission was done with improvement of vision to 20/30 plus.

Two months later, he came to the office because of severe pain above the left eye for one day. The anterior chamber was shallow on the temporal side. The tension was 35 mm. Hg (Schiøtz) and subsequently, increased to 60 mm. Hg. With eserine and pilocarpine, the tension was brought down to 20 mm. Hg (Schiøtz).

Subsequently, this tension was maintained at this pressure with Carcholin (one percent) instilled before bedtime until December 30, 1952, when he again complained of pain above the left eye. The intraocular pressure was 32 mm. Hg (Schiøtz) in the left eye. With Carcholin (one percent) morning and night, the intraocular pressure has remained around 20 mm. Hg.

His most recent gonioscopic examination was not different than that described in the previous paper.

*Gonioscopic examination.* The angle was moderately narrow above and below. There were dense pigment deposits on the posterior trabeculum. Nasally and temporally, the angles were very narrow and the posterior trabeculum could not be visualized. On the temporal side, there was a wide synechia which extended from the periphery of the iris to the cornea. On the nasal side, there were many fine peripheral synechias.

CASE 11 (6)

A. J., aged 18 years, was struck in the eye by a BB shot on January 14, 1950. Examination revealed an anterior chamber almost filled with blood but without increased intraocular pressure. In a few days, the hemorrhage was absorbed and a traumatic cataract and many keratic deposits were noted. With treatment, the condition progressed favorably until January 31, 1950. That day, he was awakened by a

very severe pain in the left eye, pain on the left side of the head and face; with nausea and vomiting.

*Examination* revealed an anterior chamber entirely filled with blood and a three-plus tension, digital. He was admitted into the Brooklyn Eye and Ear Hospital for a paracentesis and irrigation of the anterior chamber.

Following this procedure, the pain subsided and the intraocular pressure was reduced to normal levels. The cortex dissolved very slowly and the iridocyclitis continued to improve so that there were no signs of inflammation after three months.

On June 25, 1951, a discission to cut the secondary membrane and to free some encysted cortex was done at the Brooklyn Eye and Ear Hospital. Eventually his vision was 20/70. The fundus showed a diffuse choroiditis with marked pigmentary changes in the macular area.

*Gonioscopic examination.* The right eye was entirely normal. The upper angle of the left eye was wide open in spots so that the ciliary zone was seen. In the remaining area, there were peripheral adhesions of the iris to the trabecular zone with widespread iris degeneration. On the nasal side, there was a large hole in the iris with marked degeneration of the surrounding iris tissue. The angle here was narrow and there was a considerable amount of pigment on the anterior and posterior trabeculum. Below, the angle was extremely narrow, the iris atrophic with considerable dense pigment on the trabeculum. Temporally, there was considerable pigment on the trabeculum.

In the intervening two years, there has been no change in the eye.

CASE 12 (8)

E. T., a man, aged 45 years, came to the office on June 11, 1949, with a history of blurred vision in the left eye for three weeks. He had had recurrent pyelitis and cystitis for over seven years.

*Examination* revealed iridocyclitis of the left eye which cleared in a few months under local and intravenous triple typhoid therapy. At no time was the intraocular pressure elevated.

*Gonioscopy.* The first examination was on January 19, 1950. In the right eye, the angle was wide all around with a considerable amount of pigment on the posterior trabecular zone. Although the details of the left eye were hazy because of the numerous deposits on the posterior cornea, a fluffy white exudate in the lower angle could be made out, as well as considerable pigment on the entire trabeculum all around. As the eye cleared, the exudate could be seen to disintegrate and become organized to form adhesions between the root of the iris and the anterior trabeculum.

Since the previous report, he has had three attacks of iridocyclitis in the left eye. The last during February, 1953.

*Gonioscopically,* there has been a steady narrowing of the angle all around, the pigment deposits on the trabeculum have been heavier and peripheral adhesions in the lateral angles, not present earlier, are present. In spite of this, there have been no episodes of increased intraocular pressure.

## CONCLUSION

Inflammation and infections of the anterior segment of the eye cause permanent changes in the angle of the anterior chamber and these changes became progressively worse with succeeding attacks. The changes vary from small peripheral adhesions to wide synechias, exudates which may become organized to form veil-like membranes or dense cords and pigment deposits on the trabeculum, particularly its posterior portion.

An additional observation, not noted previously, was the presence of newly formed capillaries deep in the angle (Case 1). This condition had been noted in our glaucoma clinic in several cases of severe, long-standing glaucoma.

The capillaries may lie horizontally on the iris and/or the trabeculum or run radially, seeming to arise from the iris to dip into the recess of the angle, or there may be combinations of the above.

The effect of the newly formed capillaries on the function of the angle will have to be evaluated. To date, it has had no effect on the intraocular pressure in the case reported.

I again wish to emphasize that pigment deposited on the trabeculum is not a cause of glaucoma. It is possible that, in some cases, such as essential atrophy of the iris, in which the pigment may be laid down rapidly and in large amounts, the pigment might clog the trabecular spaces and be a contributing factor in causing increased intraocular pressure. In spite of the dense deposits of pigment on the trabeculum noted in so many of my cases of iritis and iridocyclitis, no increase in intraocular pressure has been found, unless the condition is acute when increased tension is probably due to increased viscosity of the aqueous; or unless, in addition, organized exudates or many peripheral synechias are present in the angle.

Whether the angle is functioning cannot be told simply from its appearance. In a number

of cases, from the appearance alone, it would not seem possible that the aqueous could drain from the anterior chamber; yet, repeated examinations revealed no increase in intraocular pressure. Perhaps the density of the pigment on the trabeculum is an illusion and the pigment is lightly packed so that aqueous can filter through.

It may be that the angle is deeper than it seems to be and possibly because of the slowness of the process, compensatory mechanisms play an increased role. The diminished formation of aqueous because of damage to the ciliary body by inflammation may be an important factor as pointed out by Grant.[2]

This would emphasize an idea expressed by me in the previous paper,[1] that an operation to reduce function and vascularity of the ciliary body, that is, cyclodiathermia, might be tried in cases of inflammation with hypertension in which other means of therapy are unsuccessful. However, with treatment of these conditions with ACTH and cortisone, this should rarely be necessary.

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# DIURNAL TONOGRAPHIC VARIATIONS AND THEIR RELATION TO VISIBLE AQUEOUS OUTFLOW*


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## INTRODUCTION

This rather extensive introduction is intended to complete what has been published previously by other authors concerning the history of tonography.

According to a literature review by Knapp,[1] Pagenstecher[2] (1878) was the first who pointed out that massage of the eyeball is followed by a reduction of intraocular pressure. After the invention of the tonometer, this observation was confirmed by Maklakoff[3] (1893), Schiøtz[4] (1909) and Grönholm[5] (1910).

In 1911, Polak van Gelder[6] studied more thoroughly the effect of massage which Schiøtz described. She applied the tonometer repeatedly at five-second intervals and found a remarkable drop of tension in normal eyes, but a much smaller drop in the tension of glaucomatous eyes. She was the first to report that the compression experiment is useful for the early diagnosis of glaucoma.

In 1913, Schoenberg[7] independently studied the effect of massage. He applied the Schiøtz tonometer continually for periods averaging two minutes and found that compression affects glaucomatous eyes less than normal ones. He too reached the conclusion that by this method "the diagnosis in doubtful cases of incipient glaucoma might be cleared up at a stage where the ophthalmoscope, perimeter, etc., are of no avail."

The results of the latter two authors' studies are in accordance with those of Grant's[8–10] more recent tonographic investigations. They did not remain uncontested, however, in later experiments of other authors. Malling (1923)[11] found, in some glaucomatous eyes and (1938)[12] in most cases of exfoliative glaucoma, a remarkably well-reduced tension after compression. Magitot[13] (1931) did not find significant


* From the Department of Ophthalmology, College of Medicine, University of Cincinnati, Cincinnati, Ohio. This study was aided by United States Public Health Grant B-158.

differences in the response of normal eyes and of glaucomatous eyes with normalized tension. Dominguez[14] observed a considerably greater decrease of intraocular pressure after compression in an eye with secondary glaucoma due to parenchymatous keratitis, than in other glaucomatous eyes.

Other authors working on compression tests were: Knapp,[3] Bader,[15] Wegner,[16] Bailliart,[17] Perez Llorca,[18] Sugasawa,[19] but the most striking studies are these by Böck, Kronfeld and Stough, and by Thomassen.

Böck, Kronfeld and Stough[20] (1934) were the first to group the examined eyes according to their initial intraocular pressure, staggered by 3.0 mm. Hg, and to compare the normal and glaucomatous eyes within each group. They compressed the eyes with the Schiøtz tonometer for two minutes, using the 15-gm. weight. In the groups between 27 and 36 mm. Hg, the effect of compression was remarkably less in glaucomatous eyes than in normal eyes, but in the groups below 27 mm. Hg, there was no difference between normal and glaucomatous eyes.

Thomassen[21] (1946) showed that in chronic simple glaucoma, the results of the compression experiment depend widely on the diurnal variations of the intraocular pressure. The drop of tension was remarkably less during the ascending phase of intraocular pressure, in the descending phase equal and in the phase of constant intraocular pressure equal or somewhat less than in normal eyes.

In accordance with this, the original purpose of the present study, performed from January, 1953, to May, 1953, was to prove to what extent the diurnal variations of the intraocular pressure may influence the results of tonography. But, as Grant's coefficient of facility of outflow C can be taken as the reciprocal value of the resistance to the outflow of aqueous humor (Prijot and R. Weekers[22]), and as K of the tonography indicates the minute volume of the total outflow of aqueous humor, we found tonography to be a useful and simple method to analyze the diurnal variations of intraocular pressure. Our results will therefore be discussed mainly from this point of view.

The diurnal variations of intraocular pressure were first described by Maslenikow[23] (1904), later evolved into a clinical method by Köllner[24] and further evaluated by a number of other authors. The causes of the diurnal variations of intraocular pressure are controversial. The literature concerning this question is too extensive to be reviewed here. Theoretically, the following should be considered (Thiel[25]):

Increase or decrease of:

1. Production of aqueous humor
2. Resistance to outflow of aqueous humor
3. Volume of intraocular structures
4. Scleral rigidity

With the aid of tonography, points 1, 2, and 3 may be determined, but not point 4. However, even if some diurnal variations of the scleral rigidity should occur (Kleinert[26]), they do not seem to cause variations of intraocular pressure. The resultant changes of intraocular volume would be insignificant compared with the volume of aqueous humor leaving the eye within the same period and would produce a minimal change to the outflow of aqueous humor to be compensated. Point 4 may therefore be neglected.

## Method

After the diagnosis of glaucoma was confirmed or excluded, in so far as possible, by a thorough clinical examination (history, visual acuity, ophthalmoscopy, perimetry, gonioscopy and, if necessary, the darkroom or water-drinking test), the patients were admitted to the ward and a diurnal intraocular pressure curve was begun.

The measurements were taken six times a day, at 6 a.m. and 9 a.m., at noon, and at 4, 7, and 10 p.m.: the first two days by tonometry and by tonography thereafter. In all eyes, at least six tonographies were performed corresponding to a period of 24

hours. In order to test the effect of pilocarpine, the intraocular pressure (IOP) was normalized in some eyes with pilocarpine and the tonographies continued over another 12- to 24-hour period.

Every measurement was preceded by observation of the aqueous veins.

All measurements were taken by the same hand and with the same instrument, the Mueller's electrotonometer. The tonometer was applied for four minutes with readings every half minute. The results were calculated according to Grant.[27] The formulas used were:

$$C = \Delta V/(P_t \text{ avg} - P_o) \cdot t$$
$$K = C \cdot (P_o - 4)$$

How far the latter formula is correct, was recently discussed by Becker and Friedenwald.[28] The reciprocal value of C, that is the resistance to the outflow of aqueous humor, R (Prijot and R. Weekers[22]) was also calculated:

$$R = 1/C$$

In order to prevent or at least diminish the possibility of error, in the measurements on one and the same eye, the following precautions were observed:

1. The tonometer was thoroughly cleaned and dried, carefully calibrated and applied vertically on the center of the cornea.

2. Measurements within the scale range 0-3 were avoided by proper choice of weight.

3. Squeezing of the lids prior to or during the tonography was avoided.

4. Patients with variations of arterial blood pressure were excluded.

5. We tried to rule out the possibility of reactive hypertension following the compression.

1. *The electrotonometer* seems to be more sensitive to dirt on the plunger than an ordinary tonometer. Our tonometer was therefore thoroughly cleaned before every measurement. According to Askovitz,[29] relatively small variations in the voltage may produce variations in tonometer readings. Our electrotonometer was therefore adjusted immediately before every measurement and another calibration made immediately after the tonography.

It is well known that the tonometer has to be applied perpendicularly with its whole footplate on the middle of the cornea (Priestley Smith[30]). Furthermore, the eye has to remain in the same position during the whole compression; that can be achieved by using a fixation point for the fellow eye considering a possible heterophoria (Knapp[1]).

2. *Scale range.* It has been recently emphasized by R. Weekers and Prijot,[31] that the readings of the tonometer below 3 are not accurate. This range was therefore avoided.

3. *Squeezing of the lids* may influence the results of tonographies in various ways. If the patient is squeezing when the anesthetics are instilled, a kind of massage of the eyeball (Bonnefon[32]) precedes the tonography and the scale readings will generally simulate lower intraocular pressure. If the patient squeezes at the beginning of tonography, gradually relaxing his lids, the first readings simulate higher intraocular pressure (Comberg and Stoewer[33]) and a greater drop of tension during the procedure is elicited. Patients who were inclined to squeeze in spite of good anesthesia and instruction were, therefore, excluded from this study.

4. *Arterial blood pressure.* As is well known, the intraocular pressure follows every rapid change in the arterial blood pressure (Wessely,[34] Thiel,[35] and others). In this connection I should mention three patients who, during tonography, showed ventricular extrasystoles with subsequent compensatory pauses (determined by electrocardiogram). During each compensatory pause, the intraocular pressure dropped rather rapidly for 3.0 to 5.0 mm. Hg. With return to normal pulse, the previous level of the intraocular pressure was again reached step by step within three pulse beats.

By analogy, irregular variations of the arterial blood pressure influence the course of the tonography, but because of their

irregularity, they cannot be eliminated and represent a source of great error. For this reason, eyes of patients with cardiac disease were also excluded from this study.

With these precautions, the error for the C in tonographies on the same eye did not exceed 10 percent, in agreement with Kronfeld.[36] This was estimated by tonographies repeated on different days but at the same hour.

5. *Reactive hypertension.* For this study in which tonographies have been repeated on the same eye at intervals of three to four hours, it was important to prove whether a reactive hypertension might have influenced the measurements.

The intraocular pressure curve of the first two days was compared with that of the following day, when tonographies were performed. In no case was the intraocular pressure curve significantly altered by tonography.

Thomassen,[21] who similarly proved the influence of repeated compressions on the course of the pressure curve, obtained similar results. He carried out four compressions on the upper limbus in three-hour intervals, using the 15-gm. weight for two minutes.

## Results

Following the procedure already described, curves for 30 eyes of 20 patients were obtained. Both eyes of 10 patients, and 10 single eyes were measured. Of the 30 eyes, seven were normal, seven suspected of glaucoma and 16 glaucomatous. Of the glaucomatous eyes, five showed glaucoma in aphakia, four chronic congestive, five chronic simple and two* a low-pressure glaucoma.

It is difficult to determine a strict borderline of what is to be taken as pathologic in diurnal variations of intraocular pressure. Hagen[37] and Thiel[38] assumed as normal diurnal variations of 2.0 to 3.0 mm. Hg. Feigenbaum,[39] Duke-Elder[40] and others consider as pathologic, a variation greater than 5.0 mm. Hg. Von Sallmann[41] pointed out that completely normal eyes may show diurnal variations of intraocular pressure up to 10 mm. Hg, but he added that in cases of glaucoma of the fellow eye, these great variations are an expression of diminished adaptability.

In this study eyes were designated as suspected of glaucoma, which were clinically normal but showed diurnal pressure variations of more than 5.0 mm. Hg. In five of these eyes the fellow eye was glaucomatous, the other two showed a large physiologic cupping of the disc.

The results of the six tonographies performed on each eye within a 24-hour period are shown in Table 1. The eyes are arranged in numerical order, in groups according to diagnosis. The same numbers are used in the discussion and in Table 4. The results contained in the "Addition" to Table 1 pertain to measurements repeated on four eyes in the same manner at a later admission when the tonographies were started in the morning of the first day of hospitalization. The first of the six tonographies is marked with an asterisk.

In Table 2, the maximum, minimum, and average values of tension (IOP), resistance (R) and total outflow of aqueous humor (K) are collected. In Table 3, in a similar way, the diurnal amplitudes of these three factors are given. Finally, intraocular pressure, resistance, and total outflow of aqueous humor (K) are presented graphically to illustrate their variations and mutual relations.

Eyes with low-pressure glaucoma responded in every respect in the same way as normal eyes and will therefore not be discussed separately.

The relationship between intraocular pressure and resistance is evident in the

* In these two eyes, marginal cupping of the disc, nasal field defect, and a large Bjerrum scotoma were present, but the intraocular pressure did not exceed 28.3 mm. Hg, showing diurnal variations of less than 5.0 mm. Hg.

## TABLE 1

RESULTS OF SIX TONOGRAPHIES IN NORMAL AND GLAUCOMATOUS EYES, TAKEN WITHIN A 24-HOUR PERIOD

| Diagnosis | Eye No. | Fellow Eye No. | Tonographies | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | a.m. | | | | | | Noon | | | p.m. | | | | | | |
| | | | IOP | 6 K | R | IOP | 9 K | R | IOP | 12 K | R | IOP | 4 K | R | IOP | 7 K | R | IOP | 10 K | R |
| Normal | 1 | 2 | 14.2 | 1.70 | 5.0 | 14.2 | 1.42 | 6.2 | *18.5 | 1.56 | 7.7 | 16.9 | 1.30 | 8.3 | 14.2 | 1.60 | 5.6 | 13.2 | 1.93 | 4.2 |
| | 2 | 1 | 11.5 | 1.21 | 5.6 | 11.9 | 1.43 | 5.0 | *16.3 | 2.69 | 3.8 | 16.3 | 1.86 | 5.6 | 11.9 | 1.93 | 3.7 | 11.9 | 2.0 | 3.6 |
| | 3 | 17 | 22.6 | 1.99 | 7.7 | 20.1 | 1.99 | 6.7 | *23.4 | 2.08 | 7.7 | 22.6 | 1.99 | 7.7 | 20.1 | 1.72 | 7.7 | 22.6 | 1.68 | 9.0 |
| | 4 | 5 | 21.8 | 2.90 | 5.0 | 21.0 | 2.20 | 6.2 | 25.4 | 3.52 | 5.0 | 25.4 | 4.23 | 4.2 | *24.4 | 3.36 | 5.0 | 20.1 | 3.96 | 3.3 |
| | 5 | 4 | 17.7 | 3.42 | 3.3 | 16.9 | 1.95 | 5.6 | 17.7 | 2.40 | 4.8 | †13.7 | 1.68 | 5.0 | *17.7 | 2.36 | 5.0 | 16.9 | 4.32 | 2.5 |
| | 6 | — | 16.9 | 3.46 | 3.1 | *16.3 | 2.16 | 4.8 | 15.0 | 1.95 | 4.8 | 16.9 | 3.03 | 3.6 | 16.9 | 3.24 | 3.3 | 16.3 | 3.10 | 3.3 |
| | 7 | 26 | 16.9 | 3.03 | 3.6 | 14.2 | 2.66 | 3.3 | 15.6 | 2.46 | 4.0 | *16.9 | 2.70 | 4.0 | 13.7 | 2.52 | 3.3 | 12.5 | 2.04 | 3.7 |
| Suspected | 8 | 9 | 24.4 | 2.35 | 7.1 | *23.4 | 2.40 | 6.7 | 22.6 | 2.60 | 5.8 | 18.5 | 2.16 | 5.6 | 18.5 | 2.40 | 5.0 | 14.2 | 1.77 | 5.0 |
| | 9 | 8 | 23.4 | 3.20 | 5.0 | *22.6 | 2.60 | 5.0 | 22.6 | 2.60 | 5.0 | 16.3 | 1.86 | 5.6 | 16.9 | 2.16 | 5.0 | 14.2 | 1.77 | 5.0 |
| | 10 | — | 18.5 | 1.80 | 6.7 | 22.2 | 1.93 | 7.7 | *30.3 | 3.08 | 7.1 | 24.6 | 2.55 | 6.7 | 21.5 | 2.14 | 6.7 | 20.1 | 1.98 | 6.7 |
| | 11 | — | †11.0 | 2.36 | 2.7 | †14.2 | 4.70 | 1.9 | *20.1 | 3.70 | 3.6 | 18.5 | 4.10 | 2.9 | 16.9 | 2.90 | 3.7 | 14.2 | 2.48 | 3.6 |
| | 12 | 15 | *16.9 | 2.92 | 3.7 | 17.7 | 1.60 | 7.1 | 22.6 | 2.00 | 7.7 | 16.9 | 2.16 | 5.0 | 12.5 | 1.51 | 5.0 | 14.2 | 1.77 | 5.0 |
| | 13 | 16 | 19.3 | 2.50 | 5.0 | 19.2 | 2.24 | 5.6 | 21.5 | 2.56 | 5.6 | 19.9 | 2.47 | 5.2 | *19.9 | 2.60 | 5.0 | 14.2 | 1.86 | 4.8 |
| | 14 | 28 | *20.9 | 2.02 | 8.3 | 26.2 | 2.12 | 8.5 | 20.9 | 2.06 | 6.7 | 16.9 | 1.30 | 8.3 | 14.9 | 1.49 | 6.2 | 15.6 | 1.08 | 9.0 |
| Glaucoma in Aphakia | 15 | 12 | 19.2 | 1.86 | 6.7 | 27.4 | 2.30 | 8.3 | 35.9 | 2.86 | 9.4 | 23.8 | 1.72 | 9.5 | *24.6 | 2.04 | 8.3 | 19.9 | 1.82 | 7.1 |
| | 16 | 13 | 16.9 | 1.85 | 5.6 | 30.3 | 2.86 | 7.7 | 31.3 | 3.21 | 7.1 | 25.5 | 3.02 | 5.8 | *30.3 | 2.41 | 9.0 | 13.2 | 1.91 | 4.2 |
| | 17 | 3 | 23.8 | 1.63 | 10.0 | 23.0 | 2.34 | 6.7 | *27.4 | 2.32 | 8.3 | 25.5 | 1.95 | 9.0 | 23.0 | 1.87 | 8.3 | 20.7 | 1.50 | 9.0 |
| | 18 | — | †14.2 | 1.15 | 7.7 | *39.9 | 2.08 | 15.0 | 42.5 | 2.35 | 14.3 | 36.4 | 1.94 | 14.3 | 33.1 | 1.97 | 12.5 | 31.0 | 1.59 | 14.3 |
| | 19 | — | 17.7 | 1.14 | 10.0 | 17.7 | 1.26 | 9.0 | *32.0 | 2.52 | 9.0 | 25.4 | 2.00 | 8.6 | 23.4 | 1.76 | 9.0 | 25.4 | 1.76 | 10.0 |
| Congestive Glaucoma | 20 | 21 | 32.3 | 2.15 | 11.2 | 31.3 | 2.29 | 10.0 | *32.3 | 1.67 | 14.3 | 32.3 | 1.91 | 12.5 | 33.5 | 2.24 | 11.2 | 27.4 | 1.55 | 12.5 |
| | 21 | 20 | 30.3 | 2.09 | 10.8 | 23.0 | 1.87 | 8.3 | *30.3 | 1.54 | 14.7 | 29.3 | 1.90 | 11.2 | 27.4 | 2.13 | 9.0 | 26.5 | 1.76 | 10.8 |
| | 22 | — | 25.4 | 2.64 | 6.7 | 26.2 | 2.03 | 9.0 | *29.3 | 4.22 | 5.0 | 28.3 | 5.06 | 4.0 | 22.6 | 3.06 | 5.0 | 22.6 | 3.21 | 4.0 |
| | 23 | — | 26.5 | 2.23 | 8.3 | 30.3 | 3.74 | 5.8 | 26.5 | 2.60 | 7.1 | 28.3 | 2.43 | 8.3 | *27.4 | 2.90 | 6.7 | 29.3 | 2.32 | 9.0 |
| Chron. simple | 24 | — | 37.2 | 1.70 | 16.7 | 30.2 | 2.64 | 8.3 | *23.8 | 1.46 | 11.2 | 23.0 | 1.55 | 10.0 | 26.5 | 1.85 | 10.0 | 23.0 | 1.40 | 11.2 |
| | 25 | — | 36.4 | 2.91 | 9.4 | 43.9 | 2.44 | 14.3 | *42.5 | 2.69 | 12.5 | 39.9 | 2.80 | 11.2 | 33.1 | 2.46 | 10.0 | 35.4 | 2.40 | 11.2 |
| | 26 | 7 | 26.4 | 1.50 | 12.5 | 42.5 | 3.36 | 10.0 | 45.3 | 2.18 | 16.7 | *45.3 | 3.40 | 10.5 | 46.8 | 2.64 | 14.3 | 32.0 | 2.61 | 9.0 |
| | 27 | — | †9.0 | 0.65 | 7.7 | *30.3 | 1.10 | 20.0 | 25.5 | 1.24 | 14.3 | 19.2 | 0.87 | 14.3 | 15.7 | 1.18 | 8.3 | †13.2 | 1.83 | 4.3 |
| | 28 | 14 | *32.0 | 2.33 | 10.0 | 36.0 | 1.34 | 20.0 | 26.2 | 1.84 | 10.0 | 20.1 | 1.32 | 10.0 | 19.3 | 1.39 | 9.0 | 15.6 | 1.38 | 7.1 |
| Low Pressure Glaucoma | 29 | 30 | 26.5 | 3.34 | 5.6 | 26.5 | 4.08 | 4.6 | *25.5 | 3.02 | 5.8 | 26.5 | 4.27 | 4.3 | 28.3 | 5.26 | 3.8 | 23.8 | 3.58 | 4.6 |
| | 30 | 29 | 26.5 | 4.08 | 4.6 | 27.4 | 4.46 | 4.3 | *27.4 | 3.48 | 5.6 | 28.3 | 5.25 | 3.8 | 26.5 | 4.26 | 4.3 | 24.6 | 3.57 | 4.8 |
| | | | ADDITION | | | | | | | | | | | | | | | | | |
| | 12 | 15 | 14.9 | 2.52 | 3.7 | *16.9 | 2.16 | 5.0 | 16.3 | 1.34 | 7.7 | 14.9 | 1.86 | 5.0 | 14.3 | 1.77 | 5.0 | 14.2 | 1.77 | 5.0 |
| | 13 | 16 | 20.9 | 2.75 | 5.0 | *16.3 | 1.86 | 5.6 | 16.9 | 1.95 | 5.6 | 13.2 | 1.28 | 6.2 | 16.9 | 2.92 | 3.7 | 14.2 | 2.22 | 4.0 |
| | 15 | 12 | 22.6 | 3.06 | 5.0 | *38.6 | 2.66 | 11.2 | 33.5 | 2.12 | 11.7 | 23.8 | 2.60 | 6.2 | 20.7 | 2.04 | 6.7 | 19.9 | 1.69 | 7.7 |
| | 16 | 13 | 18.6 | 2.64 | 4.6 | *43.9 | 2.45 | 14.3 | 45.3 | 2.17 | 16.7 | 31.0 | 1.59 | 14.3 | 20.1 | 1.99 | 6.7 | 16.3 | 2.58 | 4.0 |

IOP—Intraocular pressure in mm. Hg
K —Total outflow of aqueous humor in mm.³/min.
R —Resistance to the outflow of aqueous humor.
* —The first of the six tonographies.
† —Abrasion of the cornea was present.
Addition—Tonographies repeated on four eyes. (See text.)

TABLE 2

THE MAXIMUM, MINIMUM AND AVERAGE VALUES OF TONOGRAPHIES ON NORMAL AND GLAUCOMATOUS EYES WITHIN A 24-HOUR PERIOD

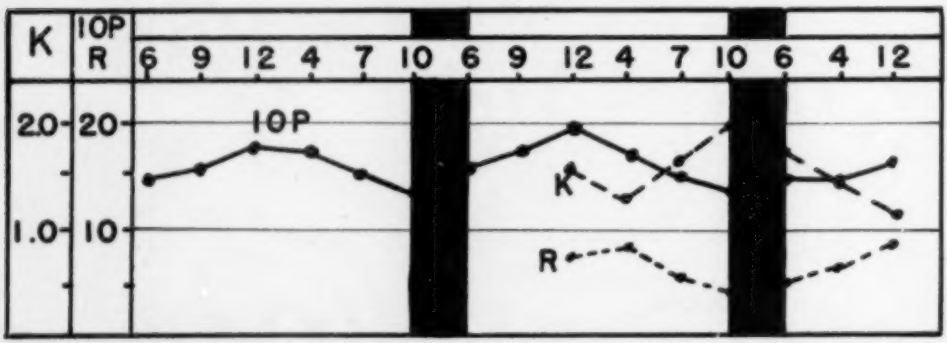
| Diagnosis | IOP | | | K | | | R | | |
|---|---|---|---|---|---|---|---|---|---|
| | Max. | Min. | Avg. | Max. | Min. | Avg. | Max. | Min. | Avg. |
| Normal | 25.4 | 11.0 | 17.6 | 4.32 | 1.21 | 2.43 | 9.0 | 2.5 | 5.0 |
| Suspected | 30.3 | 12.5 | 19.3 | 4.10 | 1.08 | 2.31 | 9.0 | 2.9 | 5.7 |
| Glaucoma in aphakia | 42.5 | 13.2 | 26.8 | 3.21 | 1.14 | 2.07 | 15.0 | 4.2 | 9.4 |
| Congestive glaucoma | 33.5 | 22.6 | 28.2 | 5.06 | 1.54 | 2.48 | 14.7 | 4.0 | 9.0 |
| Chron. simple glaucoma | 46.8 | 15.6 | 30.6 | 3.40 | 0.87 | 1.94 | 20.0 | 7.1 | 12.0 |

average values of the particular groups (table 2): with increasing resistance, the intraocular pressure rises. In Table 3, the average of the diurnal variations of the intraocular pressure and resistance show remarkable differences in the particular groups, while those of total outflow of aqueous are comparatively small. Further comparing the first three groups in this table, the normal eyes, suspected eyes and glaucoma in aphakia, neither the values of total outflow of aqueous, nor those of resistance, differ sufficiently to explain the great differences in the intraocular-pressure values of these three groups. We therefore must conclude that, for the variations of intraocular pressure, the variations of resistance and total outflow of aqueous, but still more their reciprocal relationship, must be decisive.

This can be seen from the graphs:

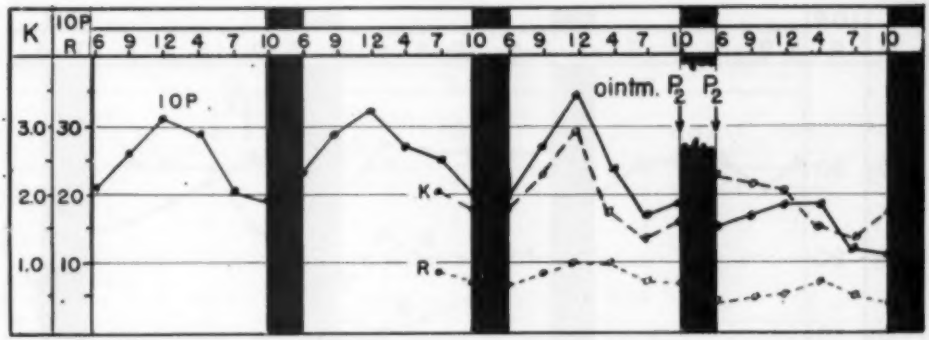
In normal eyes, as a rule, the rise of R goes together with the fall of K and vice versa (graph 1). My assumption is that in normal eyes the variations of R are not primarily existent but depend secondarily on the variations of K and "represent a compliance and adaptability of the R to variations of aqueous flow."

The R of the suspected eyes (graph 3b) and of those with glaucoma in aphakia (graphs 2 and 3a), although in normal range (below 10) and with variations of the same extent as in normal eyes, already show a course independent of K. Often the variations of K and R have the same direction, their effects on the intraocular pressure are added and result in a greater amplitude of the variations of IOP. Kronfeld's[42] com-




Graph 1* (Stepanik). Outflow, resistance, and intraocular pressure of a normal eye (Eye No. 1).

* In this and in the following graphs, the figures on the top of the graph refer to the hours of the day; the dark bars correspond to the night hours when no measurements were taken. By mistake, the intervals between the hours of the day were not properly spaced in Graphs 1 through 5.

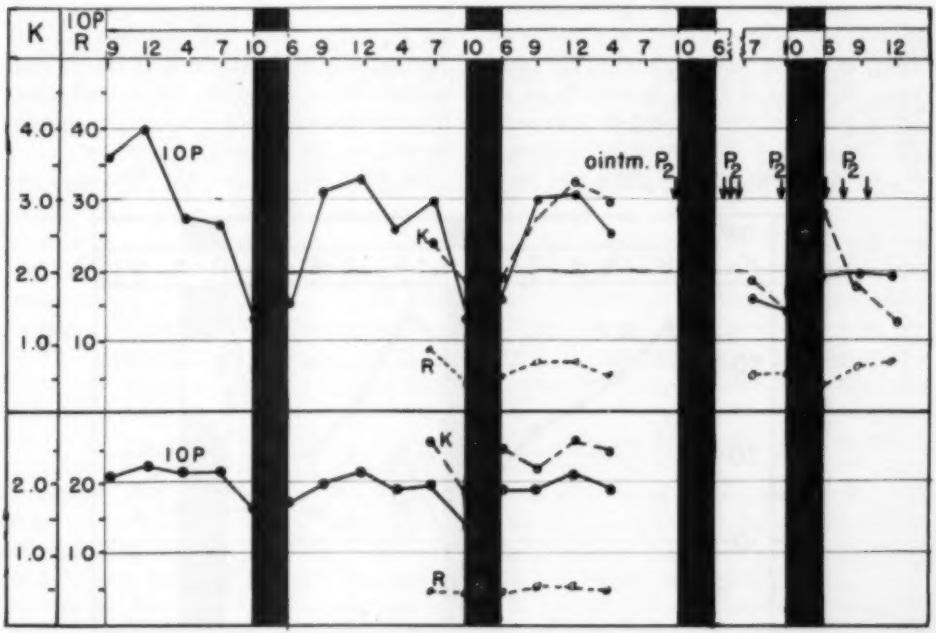



Graph 2 (Stepanik). Outflow, resistance, and intraocular pressure of a glaucomatous aphakic eye. (Eye No. 15) ($P_2$ means two-percent pilocarpine ointment.)

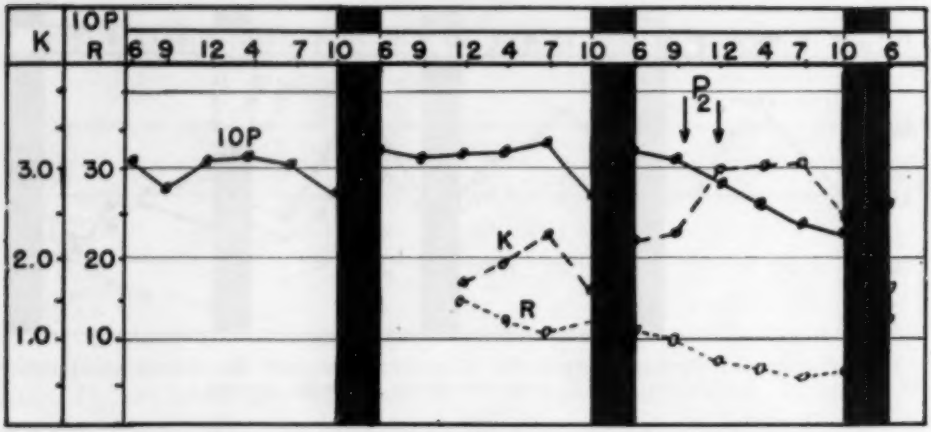
pression tests on eyes with glaucoma in aphakia, also performed on the third day of hospitalization, led to similar conclusions.

The R curve of the chronic congestive glaucoma was, throughout, within normal range in eyes No. 22 and 23; mostly in pathologic range in eyes No. 20 and 21, but in all these eyes it showed a course opposite to that of K (graph 4):

The "ability of compensation" of the R is still existent, although not to the same extent as in normal eyes, and results in comparatively small variations of IOP. The curves of these eyes, except for the level of




Graph 3 (Stepanik). Outflow, resistance, and intraocular pressure of the eyes of the same person, one glaucomatous after cataract operation, the other suspected of being glaucomatous. (Above) Glaucoma in aphakia (Eye No. 16). (Below) Glaucoma suspected (Eye No. 13, fellow eye of No. 16).



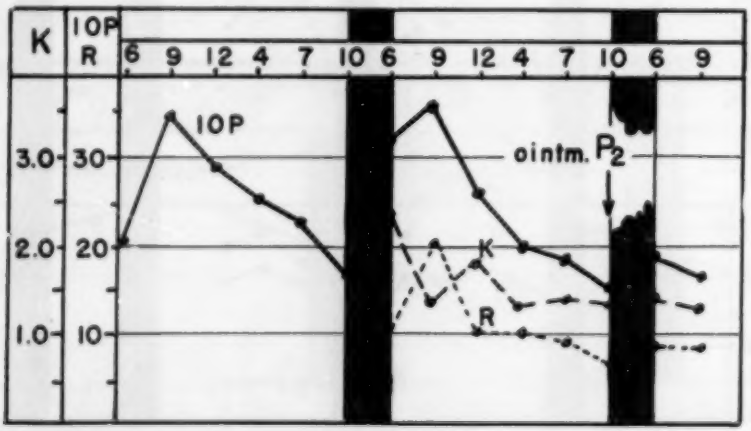

Graph 4 (Stepanik). Outflow, resistance, and intraocular pressure of an eye with congestive glaucoma (Eye No. 21).

the R, are the most similar to those of normal eyes.

The curves of the chronic simple glaucoma (graph 5) differ typically from those of the other groups:

The amplitude of the R is great; showing occasionally normal values, it rises significantly at certain times of the day. The maximum of R was at 9 a.m. in eyes 25, 27 and 28; at 6 a.m. in eye 24; and at noon in eye 26. The curve of IOP parallels the curve of R; its variations are also great and in the same direction as those of the R curve. The K curve first rises parallel to the R, but drops, as a rule, at the R and IOP maximum; then it again rises, while the R and IOP curves are dropping. Later, the K curve is again parallel to the other two curves which are gradually falling. Measurements on other days showed that this course is closely connected with the time of the day and has a regular rhythm within 24 hours as is generally known for the intraocular pressure.

In order to find possible differences be-




Graph 5 (Stepanik). Outflow, resistance, and intraocular pressure of an eye with chronic simple glaucoma (Eye No. 28).

TABLE 3

THE MAXIMUM, MINIMUM AND AVERAGE AMPLITUDE OF THE VALUES OF TONOGRAPHIES ON NORMAL AND GLAUCOMATOUS EYES WITHIN A 24-HOUR PERIOD

| Diagnosis | IOP | | | K | | | R | | |
|---|---|---|---|---|---|---|---|---|---|
| | Max. | Min. | Avg. | Max. | Min. | Avg. | Max. | Min. | Avg. |
| Normal | 5.3 | 0.8 | 3.7 | 2.37 | 0.40 | 1.34 | 4.1 | 0.7 | 2.4 |
| Suspected | 11.3 | 5.9 | 9.2 | 1.62 | 0.74 | 1.19 | 4.0 | 0.8 | 2.1 |
| Glaucoma in aphakia | 18.1 | 6.7 | 13.5 | 1.38 | 0.76 | 1.08 | 4.8 | 1.4 | 3.0 |
| Congestive glaucoma | 7.3 | 3.8 | 6.0 | 3.03 | 0.59 | 1.47 | 6.4 | 3.2 | 4.7 |
| Chron. simple glaucoma | 20.4 | 10.8 | 16.1 | 1.90 | 0.37 | 1.01 | 12.9 | 4.9 | 9.1 |

tween glaucoma in aphakia and suspected eyes, six more tonographies were performed on two eyes of each group, but this time under increased activity, beginning in the early morning the first day, immediately after the admission. The eyes with glaucoma in aphakia then resembled those with chronic simple glaucoma, while suspected eyes remained practically unchanged (addition to Table 1).

In six eyes of the three glaucomatous groups, the tonographies were continued after normalization of intraocular pressure with pilocarpine. When pilocarpine administration was started the evening before, the tonographies, as a rule, showed a lower intraocular pressure, total outflow of aqueous and resistance (graphs 2, 3 and 5). When the pilocarpine instillation was started the same day, the curves showed a remarkable fall of resistance and intraocular pressure and a simultaneous rise of total outflow of aqueous as the following measurements (graph 4).

As the intraocular pressure drops considerably, when an abrasion of the cornea occurs during a diurnal pressure curve, I was also anxious to analyze this condition. Although, with so numerous tonographies, every cornea must have undergone some kind of epithelial damage, only in four eyes could definite but small lesions of the corneal epithelium be proved by instillation of fluorescein. In these eyes, a careful tonography was performed.

The results (marked in Table 1 with a †) showed a drop of tension, in the normal eye (No. 5) a simultaneous drop of total outflow of aqueous with unchanged resistance and in eyes of the other groups (11, 18, 27) a simultaneous drop of the resistance with unchanged total outflow of aqueous. The values of these tonographies were not included in the calculations of Table 2 and Table 3.

### OBSERVATIONS ON AQUEOUS VEINS

In 13 eyes, the observations of aqueous veins supplemented the tonographic measurements: Of these eye, four were normal, two were suspected of having glaucoma, two had glaucoma in aphakia, two chronic congestive glaucoma and three chronic simple glaucoma.

In the aqueous veins of all eyes, with the exception of one, Eye 9, variations in the content of aqueous and blood, in the velocity of flow and in influx phenomena were observed, demonstrating a relationship to the K curve. With respect to the difficulty of this subject, in each eye only the one vessel group which appeared best suited for this study was followed up.

The observations made on the aqueous veins of Eyes 20 and 15 will be described in detail; those of the other eyes are schematically summarized in Table 4.

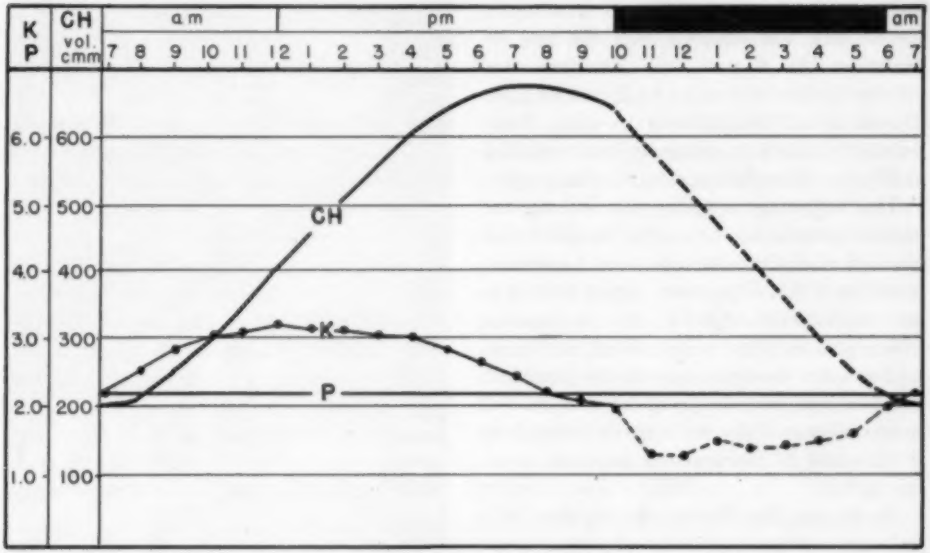
In Eye 20 (chronic congestive glaucoma, graph 4) one could see, near the limbus at the 4-o'clock position, a junction of three

TABLE 4
OBSERVATIONS ON AQUEOUS VEINS WITH CORRESPONDING TONOGRAPHIES

| Eye | | Tonography | | | | Aqueous Vein | | | | Influx Phenomenon | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| No. | Diagnosis | Time | IOP | K | R | Origin | Width | Color | Speed of Flow | Type | Speed |
| 2 | Normal | 6 | 11.5 | 1.21 | 5.6 | 4 o'clock | 0.035 | Pink | Slow | Aqueous | Slow |
| | | 9 | 11.9 | 1.43 | 5.0 | Limbal | | Pink | Slow | Aqueous | Slow |
| | | 12 | 16.3 | 2.69 | 3.8 | Emissary | | Clear | Fast | Aqueous | Fast |
| | | 4 | 16.3 | 1.86 | 5.6 | | | Clear | Mod. fast | Aqueous | Slow |
| | | 7 | 11.9 | 1.93 | 3.7 | | | Clear | Slow | Aqueous | Slow |
| | | 10 | 11.9 | 2.0 | 3.6 | | | Clear | Very slow | Aqueous | Very slow |
| 3 | Normal | 6 | 22.6 | 1.99 | 7.7 | 5 o'clock | 0.025 | Clear | Mod. fast | Aqueous | Fast |
| | | 9 | 20.1 | 1.99 | 6.7 | Scleral | | Clear | Mod. fast | Aqueous | Fast |
| | | 12 | 23.4 | 2.08 | 7.7 | Emissary | | Clear | Mod. fast | Aqueous | Fast |
| | | 4 | 22.6 | 1.99 | 7.7 | | | Clear | Mod. fast | Aqueous | Fast |
| | | 7 | 20.1 | 1.72 | 7.7 | | | Clear | Slow | Aqueous | Slow |
| | | 10 | 22.6 | 1.68 | 9.0 | | | Pink | Slow | Aqueous | Very slow |
| 6 | Normal | 6 | 16.9 | 3.46 | 3.1 | 5 o'clock | 0.035 | Clear | Fast | | |
| | | 9 | 16.3 | 2.16 | 4.8 | Limbal | | Pink | Slow | | |
| | | 12 | 15.0 | 1.95 | 4.8 | Loops | | Pink | Slow | none | |
| | | 4 | 16.9 | 3.03 | 3.6 | | | Clear | Mod. fast | | |
| | | 7 | 16.9 | 3.24 | 3.3 | | | Clear | Mod. fast | | |
| | | 10 | 16.3 | 3.10 | 3.3 | | | Clear | Mod. fast | | |
| 7 | Normal | 6 | 16.9 | 3.03 | 3.6 | 7 o'clock | 0.050 | Clear | Fast | Blood | Very slow |
| | | 9 | 14.2 | 2.66 | 3.3 | Limbal | | Clear | Mod. fast | Blood | Slow |
| | | 12 | 15.6 | 2.46 | 4.0 | Loops | | Clear | Mod. fast | Blood | Slow |
| | | 4 | 16.9 | 2.70 | 4.0 | | | Clear | Mod. fast | Blood | Slow |
| | | 7 | 13.7 | 2.52 | 3.3 | | | Clear | Mod. fast | Blood | Mod. fast |
| | | 10 | 12.5 | 2.04 | 3.7 | | | Pink | Slow | Blood | Fast |
| 24 | Chronic Simple Glaucoma | 6 | 37.2 | 1.70 | 16.7 | 2 o'clock | 0.050 | Clear | Slow | Blood | Slow |
| | | 9 | 30.2 | 2.64 | 8.3 | Limbal | | Clear | Fast | Aqueous | Slow |
| | | 12 | 23.8 | 1.46 | 11.2 | Loops | | Clear | Slow | Blood | Mod. fast |
| | | 4 | 23.0 | 1.55 | 10.0 | | | Pink | Slow | Blood | Mod. fast |
| | | 7 | 26.5 | 1.85 | 40.0 | | | Pink | Mod. fast | Blood | Slow |
| | | 10 | 23.0 | 1.40 | 11.2 | | | Pink | Slow | Blood | Mod. fast |
| 25 | Chronic Simple Glaucoma | 6 | 36.4 | 2.91 | 9.4 | 4 o'clock | 0.035 | Clear | Mod. fast | Aqueous | Slow |
| | | 9 | 43.9 | 2.44 | 14.3 | Limbal | | Pink | Slow | Blood | Slow |
| | | 12 | 42.5 | 2.69 | 12.5 | Loops | | Pink | Mod. fast | Aqueous | Slow |
| | | 4 | 39.9 | 2.80 | 11.2 | | | Clear | Mod. fast | Aqueous | Slow |
| | | 7 | 33.1 | 2.46 | 10.0 | | | Clear | Slow | Aqueous | Very slow |
| | | 10 | 35.4 | 2.40 | 11.2 | | | Pink | Slow | Blood | Very slow |
| 28 | Chronic Simple Glaucoma | 6 | 32.0 | 2.33 | 10.0 | 9 o'clock | 0.035 | Clear | Mod. fast | Blood | Very slow |
| | | 9 | 36.0 | 1.34 | 20.0 | Limbal | | Pink | Slow | Blood | Mod. fast |
| | | 12 | 26.2 | 1.84 | 10.0 | Loops | | Clear | Mod. fast | Blood | Very slow |
| | | 4 | 20.1 | 1.32 | 10.0 | | | Pink | Slow | Blood | Slow |
| | | 7 | 19.3 | 1.39 | 9.0 | | | Pink | Slow | Blood | Slow |
| | | 10 | 15.6 | 1.38 | 7.1 | | | Pink | Slow | Blood | Mod. fast |
| 22 | Congest. Glaucoma | 6 | 25.4 | 2.64 | 6.7 | 6 o'clock | 0.035 | Clear | Slow | Blood | Fast |
| | | 9 | 26.2 | 2.03 | 9.0 | Limbal | | Clear | Slow | Blood | Fast |
| | | 12 | 29.3 | 4.22 | 5.0 | Emissary | | Clear | Fast | Blood | Very slow |
| | | 4 | 28.3 | 5.06 | 4.0 | | | Clear | Fast | Blood | Very slow |
| | | 7 | 22.6 | 3.06 | 5.0 | | | Pink | Mod. fast | Blood | Slow |
| | | 10 | 22.6 | 3.21 | 4.0 | | | Pink | Mod. fast | Blood | Slow |
| 16 | Glaucoma in Aphakia | 6 | 16.9 | 1.95 | 5.6 | 11 o'clock | 0.035 | Pink | Slow | Blood | Slow |
| | | 9 | 30.3 | 2.86 | 7.7 | Limbal | | Clear | Mod. fast | Aqueous | Slow |
| | | 12 | 31.3 | 3.21 | 7.1 | Loops | | Clear | Fast | Aqueous | Mod. fast |
| | | 4 | 25.5 | 3.02 | 5.8 | | | Clear | Mod. fast | Aqueous | Mod. fast |
| | | 7 | 30.3 | 2.41 | 9.0 | | | Clear | Slow | Aqueous | Slow |
| | | 10 | 13.2 | 1.91 | 4.2 | | | Pink | Very slow | Blood | Slow |
| 10 | Suspected Glaucoma | 6 | 18.5 | 1.80 | 6.7 | 2 o'clock | 0.025 | Pink | Slow | Aqueous | Very slow |
| | | 9 | 22.2 | 1.93 | 7.7 | Limbus | | Clear | Mod. fast | Aqueous | Slow |
| | | 12 | 30.3 | 3.08 | 7.1 | | | Clear | Fast | Aqueous | Mod. fast |
| | | 4 | 24.6 | 2.55 | 6.7 | | | Clear | Mod. fast | Aqueous | Slow |
| | | 7 | 21.5 | 2.14 | 6.7 | | | Clear | Slow | Aqueous | Slow |
| | | 10 | 20.1 | 1.98 | 6.7 | | | Pink | Slow | Aqueous | Very slow |

vessels, two of which were blood vessels coming from the subconjunctiva, the third, in the middle, was an aqueous vein. It was very short (0.1 mm.) and could be recognized only when filled with blood due to a blood influx produced by compression of the recipient vein (fig. 1B).

The vein into which these three vessels emptied was 0.07-mm. wide, showed a radial course and had two aneurysms, measuring 0.4 by 0.8 and 0.7 by 1.2 mm.; in this vessel the aqueous humor produced a central lamina which could be followed into the first aneurysm (fig. 1A).




Graph 6 (Stepanik). Volume changes of choroidea. (CH) corresponding to the variations of the total outflow of aqueous (K) of Eye No. 16, when production of the aqueous (P) is presumed to be constant.

The width of this lamina, as well as its length in the first aneurysm showed the following variations during the day:

The aqueous lamina became larger from noon to 7 p.m., the ratio blood : aqueous humor: blood progressed from 1:1:2 to 1:2:2, and its length within the first aneurysm increased from one third to two thirds of the length of the aneurysm. At 10 p.m., the aqueous lamina was very thin (2:1:3) and disappeared after entering the first aneurysm.

Next morning at 6 and at 9 a.m., an aqueous lamina of similar width as in the previous afternoon could be seen (1:2:2) and could be followed as far as the middle of the first aneurysm. At 10 a.m. and at noon, two-percent pilocarpine solution was instilled. At noon and 4 p.m., the aqueous lamina was very wide (1:2:1) and could be followed throughout the first aneurysm and from time to time into the beginning of the second aneurysm. At 7 p.m., the picture was similar, but the aqueous lamina no longer reached the second aneurysm. At 10 p.m., the lamina again became narrower (1:1.5:2) and could be followed into the middle of the first aneurysm. The next day at 6 a.m., the eye was slightly irritated.

Eye 15 (glaucoma in aphakia, graph 2 and figure 2) showed an aqueous vein of 0.05-mm. width which took its origin from several limbal capillary loops at the 7-o'clock position. The recipient vein, 0.07-mm. wide, came from a scleral emissary at the 6-o'clock position. During the day, the following variations in aqueous flow could be observed:

At 7 and 10 p.m. and 6 a.m., the stream in the aqueous vein was oscillating and spontaneous blood influx (fig. 2A) occurred from time to time. At 9 a.m., the aqueous vein was clear, the current moderately fast and the recipient vein was not stratified (fig. 2B); during its compression, a blood influx occurred into the whole aqueous vein and all its branches. At noon, the aqueous vein was clear, the flow definitely faster and the recipient vein showed a short but distinct stratification (fig. 2C). The blood influx was

incomplete (slower and less pronounced), blood filled the aqueous vein but not its branches. At 4 p.m., the situation was similar to that of 9 a.m. At 7 and 10 p.m., it was as on the previous evening. Two-percent pilocarpine ointment was instilled at 10 p.m., immediately after the tonography.

The following morning, at 6 a.m., the aqueous vein was clear and the recipient vein showed a distinct stratification (aqueous: blood as 1:4). The blood influx was slow and incomplete. At 6:15 a.m., two-percent pilocarpine solution was instilled. At 9 a.m. and at noon the aqueous vein was stratified, the flow was fast, the recipient vein showed three laminae (blood : aqueous : blood as 2:1:3) and its compression produced aqueous influx.

At 4 p.m., the flow in the aqueous vein was definitely slower, the aqueous vein only slightly stratified and slow blood influx occurred during compression. At 7 p.m., the aqueous vein was clear, but the flow was slow and from time to time oscillating. At 10 p.m., the aqueous vein was filled with blood, no flow could be seen, only from time to time did a slow wave of aqueous briefly clear the aqueous vein.

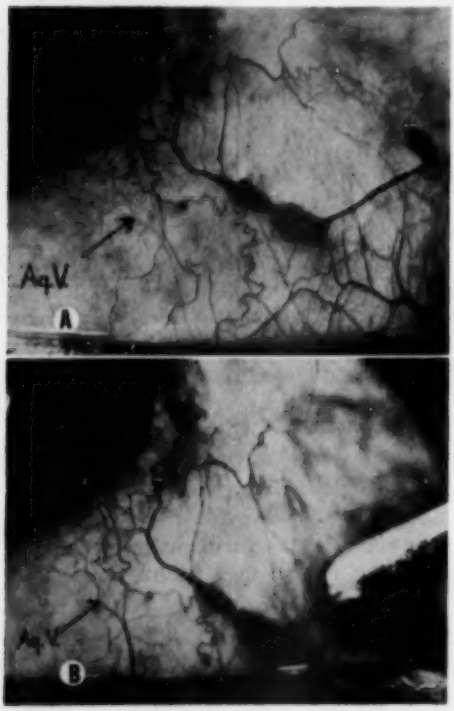


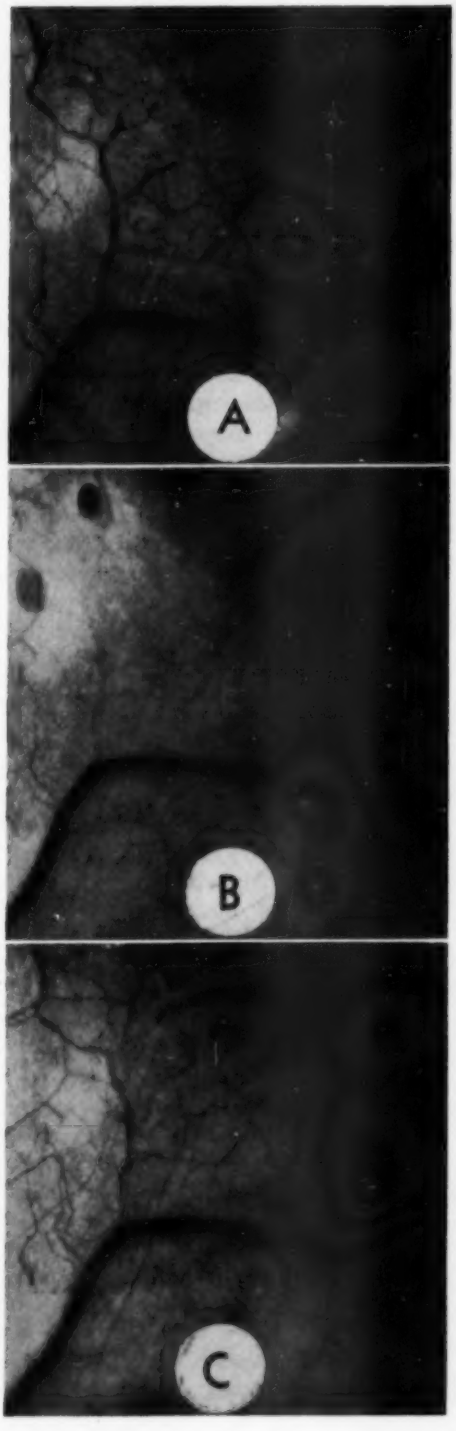

Fig. 1 (Stepanik). (A) Stratified vessel and aneurysm of Eye No. 20. (B) Blood influx after compression. The short aqueous vein, previously clear, becomes filled with blood.

The following observation on Eye 15 shows how the flow in aqueous veins depends on total outflow of aqueous. The patient came to a clinical follow up at 10 a.m. after two-percent pilocarpine solution had been instilled into his eye at 6 a.m. The aqueous vein was clear, aqueous flow fast, vena recipiens definitely stratified; its compression was accompanied by a rapid aqueous influx.

Tonography following this observation showed the intraocular pressure to be 29.3 mm. Hg; total outflow of aqueous, 2.95; and resistance, 7.1. Due to this tonography, the intraocular pressure was reduced to 19.9 mm. Hg.

An observation of the aqueous vein immediately after tonography showed that the aqueous flow was reduced and compression of the recipient vein was accompanied by blood influx. Additional compression with the electrotonometer was performed until the intraocular pressure was reduced to 17 mm. Hg. After this, the aqueous vein showed an oscillating flow and from time to time, spontaneous blood influx.

If we assume that resistance (R) was constant during this observation, then we may calculate the total outflow of aqueous (K) corresponding to every intraocular pressure (IOP) before and after compression; after the first compression a K of 1.81, after the second compression a K of 1.41. Thus the rapid aqueous influx before the tonography corresponds to a K of 2.95, the blood influx after the tonography to a K of 1.81 and the spontaneous blood influx after the second compression to a K of 1.41.




## Discussion

In chronic simple glaucoma, our measurements show that, in one and the same eye, the resistance to the outflow of aqueous humor may vary considerably. Although these eyes were pathologic, in every eye normal values for resistance were also obtained. A single tonography, therefore, cannot be decisive for the diagnosis of chronic simple glaucoma.* Since the highest values of resistance corresponded to the peak of the diurnal pressure curves, it might be advisable, in every case, first to perform a diurnal pressure curve and then tonographies at the times of day when the highest intraocular pressures were found. Only if the resistance is normal at such times, can chronic simple glaucoma be excluded with certainty.

Likewise, an eye with chronic simple glaucoma should be considered well controlled when tonographies, performed as a clinical recheck, also at the times corresponding to highest intraocular pressure readings, give normal values for resistance. It frequently requires a cumulative medication before and during this period to normalize the resistance, whereas, during the rest of the day, the resistance may remain normal, without requiring additional medication.

In a few eyes, we could conclude from repeated curves that increased activity of the glaucoma patient may raise the resistance to outflow. Therefore, at clinical rechecks, we may obtain higher values for resistance than in tonographies performed during hospitalization; additional medication may be necessary to control these eyes sufficiently.

In all eyes, variations of total outflow of aqueous could be found, which, in addition to the variations of resistance, should be considered to cause the diurnal variations of

* A similar statement made by deRoetth and Knighton[31] may be based on the same fact.

Fig. 2 (Stepanik). Aqueous vein of Eye No. 15. (A) 7 p.m., (10 p.m., 6 a.m.) spontaneously filled with blood. (B) 9 a.m. (4 p.m.) clear. (C) At noon. Clear, vena recipiens showing stratification.

intraocular pressure. In normal eyes, they were compensated by contrary variations of resistance, in chronic congestive glaucoma similarly. In suspected eyes and in glaucoma in aphakia, the resistance seemed to lose this adaptability. As a result, in these eyes, the variations of total outflow of aqueous were a decisive factor for the diurnal variations of intraocular pressure.

In chronic simple glaucoma, a remarkable depression of total outflow of aqueous was noticed when resistance and intraocular pressure reached their maxima. In these eyes, therefore, a factor should be considered, which causes an increase of resistance and simultaneously reduces the minute volume of outflow of aqueous humor. This should not be explained as a simple retention.

We know from Wessely's[43] animal experiments that a small increase of the intraocular contents raises the intraocular pressure considerably (for example, injection of 6.0 cmm. of saline into the anterior chamber produces an increase from 25 to 35 mm. Hg).

It is questionable whether we are permitted to make comparison between animal and human eyes. But, assuming some kind of accordance, we may conclude that, also in human eyes, the amount of aqueous which has to be retained to produce an increase of intraocular pressure, might be small, compared with the amount of aqueous leaving the eye during the same period of time, and that, therefore, the total outflow need not be significantly altered by this retention.

In addition, the outflow curves contribute to the understanding of the variations of visible aqueous outflow in aqueous veins.

Ascher[44] (1944) mentioned that, "in some eyes, the aqueous veins may show changes paralleling the course of the glaucoma. Filling of a formerly clear aqueous vein with more red cells may coincide with, or even announce, an increase of intraocular pressure."

In agreement with this, Thomassen[45] (1947) observed, in eyes with chronic simple glaucoma, increase of blood and decrease of clear content in aqueous veins with increasing intraocular pressure and the opposite with decreasing intraocular pressure.

My study confirmed these observations. However, in some eyes, an increase of aqueous flow coincided with increasing intraocular pressure and a decrease with the decreasing intraocular pressure.

According to Ascher,[46] the variations of the flow in the aqueous veins are connected with variations of resistance in the outlets; according to Thomassen,[47] with variations of the episcleral venous pressure. Ascher and Spurgeon[48] add that, "As the resistance increases, either the intraocular pressure goes up or the outflow rate goes down or both" and explain the reduction of outflow by the assumption of a transient or permanent narrowing of the canal outlets.

In this study, a definite relationship was evident between the visible outflow and the total outflow of aqueous. The values of total outflow of aqueous in the diurnal curve increase and decrease in the same sense as the amount of visible aqueous humor in the aqueous veins.

The most likely explanation for these remarkable variations in aqueous outflow would be variations in the production of aqueous humor. But, this cannot be definitely proved by any of the present observations. Higher K, greater outflow of aqueous humor through the aqueous veins, as well as increased flow through the anterior chamber (Lindner,[49] Langley and MacDonald[50]) would also occur if we assume a gradual increase in the volume of intraocular tissues, for example, the choroid, with production of aqueous humor remaining constant. By swelling of the choroid, aqueous humor would be displaced and would leave the eye through the regular pathways, giving the illusion of an increased production.

To what extent the choroid would have to increase in volume to produce, for example the K variations of Eye 16, is shown in Graph 6.

As a starting point, we assumed that the K of 7 a.m. was exclusively due to aqueous production at that time. The volume of the choroid (CH) is established at 200 cmm. (Becker and Friedenwald[28]). We now assume the production (P) to be constant. The K increases quickly in the first hours. At 8 a.m., it is increased by 0.3, which means that in the same time the uvea ought to increase its volume constantly by 0.3 cmm./min. to produce this increase in aqueous outflow by displacement of aqueous from the space of the vitreous. At 9 a.m. (K 2.8) it ought to increase by 0.6 cmm./min., and so forth.

In other words, the difference between the value of the assumed constant production and the respective K would be equal to the increase of uvea volume per minute.

Correspondingly, the choroid of Eye 16 would increase in volume during the day, reaching its greatest volume at 8 p.m., when the K is again equal to the assumed production and would then gradually decrease in volume (corresponding to a K smaller than the production 8 to 10 p.m. and 6 to 7 a.m.). The supposed course of K from 10 p.m. to 6 a.m. is shown shaded. As one can see from the graph, the choroid would have to increase, in this case, from 200 to 671 cmm.

Whether the variations of the K curve are due to variations of aqueous production or of volume of the intraocular tissue or due to a combination of both, cannot as yet be determined.

## Summary

In each of 30 eyes—seven normal, seven with suspected glaucoma, and 16 glaucomatous—six tonographies were performed within a 24-hour period. The diurnal variations of intraocular pressure (IOP), of resistance to outflow of aqueous humor (R), and of total outflow of aqueous humor (K) were determined.

In this study, resistance was, on the average, higher in glaucomatous eyes than in normal or suspected eyes. In chronic simple glaucoma resistance was mostly near the borderline, but showed definite pathologic increase at the times of the peaks of the pressure curve and normal values when the diurnal pressure curve was near its minimum. Therefore, single tonographies cannot with certainty exclude chronic simple glaucoma. It is advisable first to make a diurnal pressure curve and to study tonographically at such times of the day which correspond to the peaks of the pressure curve.

In all eyes, variations of the total outflow were found. The total outflow of eyes with chronic simple glaucoma was, on the average, smaller, with lower diurnal variations, than the average total outflow of the other groups; as a typical finding, a depression of the outflow coincided with the peak of resistance and intraocular-pressure curve.

To produce diurnal variations of intraocular pressure, the variations of resistance and outflow were decisive. Due to the fact that variations of resistance and outflow exert opposite effects on intraocular pressure, the mutual relation of these figures is of paramount importance.

Within the first hours after instillation, pilocarpine produced a drop in the resistance and in the intraocular pressure and simultaneously an increase of total outflow. Later on, a decrease of all three factors was observed.

The variations of visible outflow of aqueous humor observed in aqueous veins were in the same direction as the variations of total outflow.

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# THE MECHANICAL AND NEUROVASCULAR SCHOOLS IN THE MECHANISM OF PRIMARY GLAUCOMA*

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In the past few years considerable emphasis has been placed by reviewers in the field of glaucoma on a continuing alleged conflict between two antagonistic schools of thought regarding the mechanism of primary glaucoma; namely, the followers of a neurovascular theory and those who accept a mechanical theory.

I believe the time has arrived for considering the validity of such a conflict and for presenting a reasonable concept of the primary glaucomas which will help in doing away with the idea of the two schools of thought.

The neurovascular school is presented as believing that all primary glaucoma arises from a disturbance in the ocular neurovascular system, while the mechanical school is presented as considering primary glaucoma chiefly from a mechanical point of view.

I will agree that there is a point of view, championed by Duke-Elder, that considers all primary glaucoma, both acute congestive and simple glaucoma, to be of neurovascular origin. However, the viewpoint that there is or has been a school of thought which considers all primary glaucoma to be mechanical in origin is untenable.

Scheie[1] considers the mechanical school to be synonymous with that outlined by the followers of the gonioscopic classification of primary glaucoma. Since I have been espe-

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cially interested in that classification, I feel compelled to refute the idea that those men who have been instrumental in its evolution had any purely mechanistic theory of the origin of primary glaucoma, nor did they at any time consider simple glaucoma on a mechanical basis.

The gonioscopic group, as a result of observations on the chamber angle, concluded that there are two separate disease entities included in adult primary glaucoma. Their mechanical concept is concerned *only* with the immediate angle-blocking mechanism of acute glaucoma, without denying other more remote ultimate causes such as the neurovascular ones.

Let us now reconsider the subject of the actual controversy. It resolves itself into a conflict between Duke-Elder's group and the so-called "gonioscopic group" in the United States, particularly in regard to a portion of the mechanism of acute narrow-angle glaucoma. No conflict exists in regard to simple glaucoma.

Duke-Elder considers acute narrow-angle glaucoma to be due fundamentally to a recurrent circulatory instability which is the result of neurohumoral and endocrine disturbances, as well as upsets in the sympathetic-parasympathetic balance. These lead to capillary dilatation, increased vascular permeability, stasis and local edema, and increased intraocular pressure. The vascular crises with congestion and edema of the ciliary body may thus secondarily force the iris against the trabecular wall and produce peripheral anterior synechias.

Thus the narrow-angle glaucoma, according to Duke-Elder, only *follows* repeated attacks of congestion and mild rises of tension which, at first, are without narrowness of the angle. The narrowness of the angle only serves to make the crises dangerous. Duke-Elder stated in his 1952 Los Angeles lectures that there is no doubt that congestive glaucoma is usually characterized by narrowness of the angle but a typical acute attack may occur without a narrow angle.

The "gonioscopic group," on the other hand, believes that whatever the precipitating factors, acute glaucoma can occur only where the angle is primarily narrow and so can be found *only* in anatomically predisposed individuals.

Duke-Elder considers the rise in tension in congestive glaucoma which may occur toward the end of the day to be a manifestation of a disturbance in the regulatory mechanism of ocular pressure. Such instances may, however, be explained from the viewpoint of the gonioscopist, on the basis of iris block associated with dilation of the pupil at the time of day when light stimuli decrease.

It is obvious that the emotional factors which may precipitate the sharp rise in tension act through a neurovascular medium. But the two groups differ in how the emotional factors act.

According to Duke-Elder, the emotional factors disturb the regulatory mechanism, causing a rise in tension in the manner already described. The gonioscopist feels that the emotional factors act on the vascular elements directly, producing iris block *only in predisposed eyes with narrow angles.* There is no attempt on the part of the followers of this concept to deny the importance of neurovascular factors or psychic stimuli as the ultimate causes of the angle blocking. In fact, in many cases of this disease these factors are rather obvious.

It is apparent that the crux of our conflict is whether the angle narrowness is a primary or only a secondary factor. What evidence do we have of the relation between primary angle narrowness and acute glaucoma? This may be considered under two headings:

1. The clinical evidence that *local* obstruction of chamber angle drainage may result in a sharp increase in intraocular pressure.

2. The evidence that acute glaucoma is due to a primary narrowness of the chamber angle.

The clinical evidence that local obstruction

of the angle drainage may lead to elevated intraocular pressure falls under three headings: (1) The secondary glaucoma following cataract operation, (2) mydriatic glaucoma and glaucoma associated with intumescent cataract, and (3) pupil-block glaucoma.

The secondary glaucoma following operation for cataract in previously nonglaucomatous eyes has been shown[2, 3] to be due to blocking by peripheral anterior synechias, usually as a result of collapse of the anterior chamber which mechanically places the iris in contact with the trabecular wall.

Glaucoma following mydriasis is a perfect example of a locally and mechanically induced glaucoma. Most of us, especially before the days of chamber depth consideration, have observed cases of acute glaucoma following instillation of mydriatics, usually for refraction. We are all also familiar with the positive darkroom test which can reasonably be thought of as a mechanical dilatation of the pupil.

In 1941,[4] I made a study of the effects of provocative dilatation of the pupil with mydriatic drugs on 20 eyes with shallow chambers to obtain evidence for the mechanical causation of acute glaucoma. The series of 20 eyes consisted of 13 nonglaucomatous eyes of patients with unilateral acute glaucoma in the other eye and seven acute glaucomatous eyes which had become normalized with miotics. In seven of the 20 eyes from both groups of cases, the tension rose to a high level, and afterward was reduced to normal by the use of a miotic drug. Grönholm,[5] in 1910, had a case in which he was able to produce a similar tension rise or fall at will with mydriatic or miotic drugs.

It is worth observing, in contrast, that at no time has dilatation of the pupils in patients with simple glaucoma given any significant rise in ocular tension.

Further evidence of mechanical provocation is obtainable from the study of patients with acute glaucoma resulting from the intumescence of cataractous lenses. In these cases, the shallowing of the anterior chamber by the increased lens volume sets the stage for the same mechanical closure of the chamber angle that we find in acute glaucoma. In two such instances, I have observed the onset of acute glaucoma precipitated by mydriasis.

A third evidence that local mechanical narrowing and blockage of the angle may produce acute glaucoma is that resulting from either true or relative pupillary block. Here, as a result of inability of aqueous to pass from the posterior to the anterior chamber, the iris is pressed forward by the aqueous, which continues to form and narrows the chamber angle, finally blocking it.

True pupillary block occurs in inflammatory iris bombé while relative pupil block occurs in air blockade. That relative pupil block also occurs in acute primary glaucoma has been shown through the effect of peripheral iridectomy in early cases.

Scheie and Frayer[6] and Barkan[7] found that air injected into the anterior chamber may cause an acute rise in ocular tension due to valvelike obstruction between the pupil border and anterior iris and the posterior surface of the air bubble. This obstruction leads to an iris-bombé effect which closes the chamber angle. These authors observed that wide pupillary dilatation or iridectomy prevented the rise in tension. Friedenwald[8] attributed the glaucoma following anterior lens dislocation to the same ballvalve principle.

These types of glaucoma may be considered as evidence that local angle blockage may produce an acute rise in ocular tension.

The evidence that acute glaucoma is due to a primary narrowness of the chamber angle may be considered under three headings:

1. Measurements of the anterior chamber depth in eyes with acute narrow-angle glaucoma.
2. Gonioscopy.
3. The evidence derived from observation of the iridectomy operations, especially peripheral iridectomy.

*Measurement of anterior chamber depth* has led to the conclusion that the shallow anterior chamber in acute glaucoma can and does exist before onset of the disease (Rosengren[9]). This is also suggested by the work of Törnquist[10] who studied the anterior chamber depth in 45 pairs of twins with normal eyes and concluded that the chamber depth is genetically determined. He assumed that the shallow anterior chamber in narrow-angle glaucoma is also genetically determined and so is a predisposing factor.

*Gonioscopy* has shown that, during the acute congestive attack of glaucoma, the angle is blocked by iris; while in those cases in which miotics are successful in normalizing the tension, the angle becomes open though narrow. It has shown also that in cases in which the angle is blocked by synechias, basal iridectomy acts by freeing the obstruction in a portion of the angle circumference.

The work of Grant[11] with the electronic tonometer tends to add to the gonioscopic viewpoint in regard to acute narrow-angle glaucoma. In the acute phases of narrow-angle glaucoma, Grant found marked obstruction to aqueous outflow. After normalization of tension by miotics, the aqueous outflow was found to be normal except where peripheral anterior synechias had formed. Thirteen fellow eyes of eyes with acute glaucoma were examined gonioscopically and found to have very narrow but open angles. In these eyes the tension and rate of aqueous outflow were normal. In one eye an acute congestive attack later was observed during which the rate of aqueous outflow became greatly reduced, but after normalization with miotics, again became normal.

*Iridectomy.* The relatively recent work which has been done in regard to the effect of peripheral iridectomy in cases of early narrow-angle glaucoma lends further emphasis to the importance of the immediate mechanical factor in the origin of acute narrow-angle glaucoma.

Haas and Scheie[12] found that in cases of narrow-angle glaucoma, peripheral iridectomy causes deepening of the chamber angle due to relief of a physiologic iris bombé or relative pupillary block resulting from resistance to aqueous flow from the posterior to the anterior chamber between the pupil border of the iris and the anterior surface of the lens.

An interesting case reported by Haas and Scheie is worth citing as evidence that a mechanical and not a nervous mechanism is the immediate cause of acute glaucoma. This patient, showing the classical picture of narrow-angle glaucoma, showed a high tension under emotional stress or when miotics were stopped. At this time the angle became closed. A small peripheral iridectomy was made but the tension was found to be 80 mm. Hg (Schiøtz) on the following day. The site of the iridectomy had become incarcerated in the wound and was no longer patent. On miotics, the tension again fell to normal. Another peripheral iridectomy was performed. This time it remained patent. Since then the tension remained normal, even after instillation of mydriatic drugs.

How can we explain the failure of many of the European glaucomologists to see eye to eye with us on the question of iris block? I believe that the answer is suggested in the statistics of Paul Weinstein in regard to the incidence of the various component entities in the adult primary glaucoma group.

In his recent book, Weinstein[13] stated that primary glaucoma may be subdivided as follows: chronic inflammatory glaucoma, 72 percent; acute inflammatory glaucoma, 18 percent; and glaucoma simplex, 10 percent. These figures are quite at variance from the experience of ophthalmologists in the United States.

I am quite certain that the variations in the anterior-chamber depth in simple glaucoma and the absence of congestion in many incipient cases of acute glaucoma have confused the clinical separation of the two forms of glaucoma. I do not mean to imply that there are not cases where the distinction may be so

difficult that only observation over a period of time can lead to a correct diagnosis.

## Mechanism of primary glaucoma

We are now ready for a brief presentation of a concept of the mechanisms of the adult primary glaucomas.

### Simple glaucoma

Simple glaucoma is characterized by an absence of any recognizable disturbance in the chamber angle. Because of this I used the term normal angle-width glaucoma to distinguish it from narrow-angle glaucoma. Haas and Scheie[12] suggest "open angle" as a better term. I, too, now like this term better.

The site of obstruction in simple glaucoma is unknown but is believed to be at the collecting channels efferent from Schlemm's canal. The immediate cause of obstruction is probably vasoconstriction or sclerosis at this site.

It is interesting that support for the idea that vascular sclerosis as the chief immediate factor in simple glaucoma has increased. Schnabel's idea that cavernous degeneration of the optic nerve precedes cupping of the disc finds support here. In 1942, I[14] suggested a relation between vascular sclerosis and simple glaucoma based on clinical observations in patients with retinal venous obstruction and simple glaucoma. These observations were corroborated by Wessely[18] in 1947.

Duke-Elder,[16] in 1949, suggested that disc changes in simple glaucoma may be due to sclerotic changes in the nutrient vessels of the optic nerve. He described three manifestations of sclerosis: (1) If the posterior segment vessels are chiefly involved, excavation of the optic nerve results, even without abnormal tension; (2) if the anterior segment vessels are chiefly involved, a rise in tension is the early feature of the disease; while (3) if the vascular involvement affects both segments, the typical picture of simple glaucoma results.

Friedenwald,[17] in 1936, had suggested histologically that glaucoma simplex may be due to sclerosis of vessels associated with Schlemm's canal which he considered to be afferent to the canal.

Disturbances of the venous pressure in the vessels leading from Schlemm's canal has been postulated recently as the immediate cause of simple glaucoma. Thomassen and Bakken[15] found that before aqueous veins emptied into intrascleral blood-containing veins, the two vessels for a distance ran side by side in a rigid scleral canal. During this portion of their course the two fluids are separated from each other only by the vein wall and the layer of endothelium making up the aqueous vessel wall. Thomassen and Duke-Elder have suggested that increased venous pressure in the blood-containing vein might compress the aqueous vessel and impede the exit of aqueous.*

Poos[19] postulated that the orbital venous pressure influencing the blood flow through the vortex veins may be important in causing glaucoma.

The ultimate causes of the simple glaucomatous process have received great attention in the literature but are still very controversial. However, as important as they are, they do not affect our clinical ideas of the intraocular process. They may be considered as follows:

1. Neurovascular stimuli
   a. autonomic
   b. psychic
2. Control center disturbances
3. Humoral (?)

Magitot,[20] on the basis of determinations of acetylcholine and cholinesterase in the aqueous, postulated a regulating center for ocular tension located in the diencephalon.

* Ashton and Smith have presented evidence to show that the vessels which occasionally accompany the canal outlets within the sclera are in reality arterioles. They believe that these vessels have no significance in the control of aqueous flow. (Ashton, N., and Smith, R.: Anatomic study of Schlemm's canal and aqueous veins by means of neoprene casts: III. Arterial relations of Schlemm's canal. Brit. J. Ophth., 37:577, 1953.)

A disturbance of that center might result in a deficiency of the parasympathetic system which leads to localized edema and hemostasis which in the eye causes glaucoma.

Schmerl and Steinberg[21] believe that the posterior lobe of the pituitary secretes two active principles, one of which causes an increase and the other a decrease in intraocular pressure. These are secreted into the spinal fluid and carried to a diencephalic center which controls the intraocular pressure through the autonomic nerves to the eyeball. They believe that the parasympathetic system transmits the stimuli which increase the intraocular pressure while the sympathetic pathways transmit the stimuli which lower the intraocular pressure. Elwyn,[22] Filbry,[23] and Pau[24] postulate a deficiency of normal parasympathetic control over the intraocular pressure.

### Acute narrow-angle glaucoma

Acute narrow-angle glaucoma is characterized by a single salient feature; namely, narrowness of the chamber angle. Certain anatomic factors may be the cause of the narrowing:

1. Shortness of the eyeball associated with high hyperopic errors of refraction.
2. Continuous growth of the lens throughout life.
3. Smallness of the cornea.
4. The thickness of the iris.

Certain physiologic factors may be superimposed to produce the blocking of the already narrow angle:

1. Accommodation.
2. Pupillary dilatation.
3. Ciliary body congestion.
4. Relative pupillary block or physiologic iris bombé.

These physiologic factors may be considered to be the immediate causes of angle block.

The only factor which may be considered as purely mechanical is dilatation of the pupil. The others may better be considered as of neurovascular origin. All of them lead to mechanical iris block. Scheie suggests that physiologic iris bombé may be the result of an increase in the rate of aqueous flow, whether on an emotional or other basis. This would balloon forward the iris periphery until it comes in contact with the corneoscleral wall.

Grant's work, however, indicates that there is no increase in production of aqueous here. The mechanism might be explained by nerve stimuli causing a proper combination of factors such as vasodilation in the ciliary body and contraction of the ciliary muscles, allowing the lens to move forward slightly. The resulting increase in relative pressure behind the iris over that in front causes the "physiologic" iris bombé.

The ultimate causes of angle obstruction may be either neurovascular or humoral stimuli. The neurovascular stimuli may be autonomic or psychogenic in origin.

There is an argument frequently presented which is used to imply that there is no real distinction between acute narrow-angle and simple glaucoma. This argument includes the observations that psychic factors affect both types of glaucoma and that miotics are effective in reducing the tension in both. As stated previously, psychic factors may be the neurovascular precipitants of acute glaucoma and also have an important influence on the neurovascular factors in simple glaucoma. The presence of psychic factors in both types of glaucoma is associated with different intermediate mechanisms.

Although we do not know exactly how the miotics work in simple glaucoma, we believe that these drugs have a vascular effect, either in affecting the osmotic factors in the ciliary and iris vessels or the efferent vessels from Schlemm's canal.

Duke-Elder believes that the miotics cause a fall in the venous pressure in the exit veins from the eye and thus increased drainage of aqueous. In acute glaucoma, we believe that the miotics keep the narrow angle open or open the blocked angle mechanically by simple pupil-constriction.

**ACUTE NARROW-ANGLE GLAUCOMA**

SITE OF OBSTRUCTION — CHAMBER ANGLE

IMMEDIATE CAUSES OF OBSTRUCTION:

A. DILATATION OF PUPIL
B. PHYSIOLOGIC IRIS BOMBE
C. VASODILATATION IN CILIARY BODY

} IN PREDISPOSED EYE WITH NARROW ANGLE

ULTIMATE CAUSE OF OBSTRUCTION:

A. NEUROVASCULAR STIMULI
   1. AUTONOMIC STIMULI
   2. PSYCHIC STIMULI
B. HUMORAL (?)

**SIMPLE OPEN-ANGLE GLAUCOMA**

SITE OF OBSTRUCTION—PROBABLY AT COLLECTING CHANNELS EFFERENT FROM SCHLEMM'S CANAL

IMMEDIATE CAUSE OF OBSTRUCTION:
PROBABLY VASOCONSTRICTION OR SCLEROSIS AT SITE

ULTIMATE CAUSE OF OBSTRUCTION:

A. NEUROVASCULAR STIMULI
   1. AUTONOMIC STIMULI
   2. PSYCHIC STIMULI
B. CONTROL CENTER DISTURBANCE (?) HYPOTHALAMUS (?)
C. HUMORAL (?)

Fig. 1 (Sugar). Graphic representation of the two forms of adult primary glaucoma.


## Summary

In summary, the graphic representation of the two forms of adult primary glaucoma shown in the Figure 1 indicates that the mechanism of primary glaucoma cannot be thought of on the basis of conflicting mechanical or neurovascular schools of thought. Both mechanisms are really involved together in varying ways, depending on the type of glaucoma being considered. The mechanical mechanism is activated by neurovascular stimuli while neurovascular mechanisms often require some mechanical changes to be effective.


*28 West Adams (26).*

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# BOWEN'S DISEASE*

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There are quite a few synonyms for this condition, such as precancerous epitheliomatosis, intraepithelioma, carcinoma in situ but, since Bowen, in 1912, reported two cases of this lesion it is more or less commonly designated as Bowen's disease. The exciting factor may be some sort of chronic irritation, for example, exposures to various kinds of irradiations, chronic inflammation, or even trauma. However, the etiology is not always clear. It is definitely a neoplasm of variable, though low-grade, malignancy.

Within the epidermis, growth is proliferative but, ordinarily, the lesion does not metastasize. It is actually a dyskeratosis with the epidermal structure demonstrating much disorder and irregularity. Histologically there are mitotic figures, some of which are atypical. There is always potential danger of these atypical mitotic cells spreading (incidence of spread, about three percent).

The lesion usually originates at the limbus from the zone of epithelial transition. The ratio of malignancy increases from skin to mucosa, so much so that conjunctival involvement may demand enucleation.[1] Since this type of epithelium is unstable, a full-fledged squamous carcinoma may ultimately develop.

According to McGavic,[2] who reported his observation on five cases of involvement of the conjunctiva and the cornea, the lesion appears on the cornea and conjunctiva as slightly elevated, diffuse, sometimes multiple, highly vascularized patches of reddish gray, gelatinous tissue. There is always considerable inflammatory reaction beneath the new growth, sometimes enough to mask the tumor entirely.

* From the Mascati Eye Hospital.

Sometimes the condition simulates, macroscopically, certain other pathologic conditions from which it should be differentiated. It may be mistaken for Mooren's ulcer. The case of Mackie and Edwards[3] actually did start from a Mooren's ulcer. Pannus, benign epithelial proliferation, fatty degeneration of the cornea, as well as epithelial dystrophy of the cornea, present similar findings. In making a differential diagnosis, one should also keep in mind the possibility of a filtering cicatrix of the cornea, xeroderma pigmentosum, large phlyctenules, pinguecula, and pterygium. Histopathologic examination is essential in an exact diagnosis.

Treatment has been most unsatisfactory. The malignant tendency of the lesion has forced surgeons to resort to enucleation. Even exenteration of the orbit has been advocated.[4] Simple excisions, electrolysis, X-ray irradiations, and radon have been tried. Radiation sufficient to destroy the tumor has frequently damaged the intraocular structures, with disastrous sequela. Excision and electrolysis may cause the lesion to spread.

Esterman, et al.,[5] were the first to try contact irradiation, using a Chaoul tube, operating at 60 kv. under five ma., and without filter. The dose they employed was 610 r/minute for 1.5 minutes every two days, to a total of 5,400 r administered during nine treatments. The result was very good.

Fig. 1 (Mascati). Bowen's disease. Note the encroachment of the growth on the cornea.

Recently François, et al.,[6] reported disappearance of the growth following contact radiotherapy, without such ocular damage as lens opacities and so forth. They used Philips Metalix tube at 50 kv., under two ma., and without filter. They employed surface localization of 51 r for nine seconds, with a total of 5,150 r in 10 treatments. They were so much encouraged with the result, that they advocate this form of therapy even in basocellular epithelioma of the bulbar conjunctiva.

## Case report

D. H. (HP/13-D), a man, aged 60 years, was first seen by me in the out-patient department of the Mascati Eye Hospital on December 21, 1949.

*Past history.* About nine years before the patient had had sore eyes which frequently became acutely inflamed. He had treated himself with such irritants as copper sulfate, silver nitrate, dionine, and so forth. Since then, the left eye has been troublesome and becomes inflamed more often. A small growth on the left eyeball, on the customary site of a pterygium, medially, was noticed about six years ago. It did not progress. Three years ago a small growth from the under surface of the upper eyelid of the same eye, in apposition to the area of bulbar growth, was excised. The specimen was examined microscopically and the histopathologic report stated it was a papilloma of the conjunctiva with early changes characteristic of Bowen's disease. The bulbar growth was not touched at all.

*Eye examination.* When I first saw the patient, there was slight infiltration of the cornea on the medial side, and the growth (to the naked eye) looked rough and papillomatous. Situated almost at the limbus, it was partly overriding the periphery of the cornea medially. Excision and a mucous-membrane graft were advised. Due to my absence from India during the next 10 months, I could not take care of the patient. He again consulted me on my return, after excision of the growth elsewhere.

*Second examination.* The patient was admitted to the hospital on December 12, 1950. Examination on admission revealed the right eye to be normal except for sclerosis of the lens, with distance vision, 6/9. The left eye had vision of moving bodies.

The upper lid had a scar on the under bulbar surface. The extraocular muscle balance was within normal range. Intraocular pressure was 17 mm. Hg (Schiøtz). The lacrimal apparatus was normal and patent.

The conjunctiva had a papillomatous growth situated medially on the limbus extending from the 11-o'clock position to the 7-o'clock position. The superior fornix was very narrow due to previous surgery. Medially (nasally) the caruncle was as far as the growth extended.

The edges of the tumor were slightly irregular but otherwise distinctly discernible. The encroachment on the cornea was distinct and it was seen riding over the medial sector.

The lesion was of firm consistency with a rough, granulated, shining surface. It was whitish gray. One could easily pick up a portion of the growth and demonstrate its mobility which increased the farther away it was from the limbus. There were full, turgescent blood vessels running over it.

Biomicroscopy with low magnification showed a follicular arrangement. The sclera was adherent to the conjunctiva medially at the limbus below the growth. The cornea was very hazy. The tumor overrode the limbus and was adherent to the cornea. There was extensive secondary vascularization.

The anterior chamber appeared to be of normal depth. The pupil reacted to light normally and was of normal size. The iris also had normal appearance. Due to haziness of the cornea, the media and the fundus could not be properly examined.

Urinalysis and Kahn reaction were negative.

It was proposed to excise the growth to its full extent from the bulbar conjunctiva as well as from its palpebral portions, and from the cornea where a partial keratectomy would have to be done. A mucous membrane graft from the lower lip would eventually be used to cover the bare portion of the sclera and Tenon's capsule.

*Preoperative.* The left eye was atropinized. Procaine penicillin (400,000 units) was administered daily for a week. Mild sedation and vitamin C (300 mg.) three times daily were prescribed.

*Anesthesia.* Facial nerve block was done by the O'Brien technique. Retrobulbar injections were made in the muscle cone and in the region of the ciliary ganglion. Hyaluronidase (50 TRU) was incorporated with two-percent novocaine (5.0 cc.) in the facial nerve block and the same amount of the enzyme with 1.0 cc. of four-percent novocaine for retrobulbar injection. Surface anesthesia was with Pantocain (1.0 percent).

*Operation.* A superior rectus bridle suture was used. The conjunctiva from the medial side of the lower fornix right up to the superior fornix on the same side was dissected free from the underlying episcleral tissues and excised. The medial arc of the limbus was also treated in the same manner, and the conjunctiva bearing the growth was completely removed. The bleeding points were sealed with thermal cautery.

The dissection was made easier and quicker by using Gill's corneal dissector. Medially, the conjunctiva was removed up to the caruncle.

A partial keratectomy was done. The superficial layer of the epithelium, together with the topmost layers of the substantia propria, were incised with a very sharp No.-10 Bard-Parker knife. The incisions made were in the shape of a cross, meeting in the center of the cornea. With jeweller's forceps, the layer from one quadrant was picked up at the center and the opaque layer bearing the growth was radically stripped off with the aid of Gill's and Took's dissectors and Castroviejo's keratectomy corneal scissors. The cornea was kept moist all the time with drops of normal saline. The pattern of the iris and the condition of the pupil were clearly brought into view as the corneal opacity was being removed.

After the keratectomy was finished, a semilunar strip of mucous membrane was excised from the patient's lower lip. The strip was one inch long and one-half inch wide in the middle gradually tapering off at the ends. After all the glandular structures were meticulously removed from the under surface, the graft was placed on the bare surface of the exposed sclera on the medial side of the limbus. Great care was taken to form the shallowed fornices. The extreme nasal side of the graft was sutured to the border of the conjunctiva adjacent to the caruncle with 6-0 silk. Wherever possible the other borders of the graft were sutured with the same material to healthy conjunctiva. If there was no healthly conjunctiva, the graft was sutured directly to the underlying sclera, using a fine Grieshaber sclerocorneal needle.

*Postoperative.* After dressing, both eyes were bandaged to minimize movements. The postoperative period was without incident except for a mild iritis which developed after about two weeks and was due to removal of the bandages before complete healing of the cornea had taken place. This was at once observed with fluorescein. The eye was bandaged for another week and nonspecific protein therapy was instituted. The iritis was cured. Postoperatively, a two-percent solution of atropine-sulfate and one-percent aureomycin ointment were used. There was a remarkable improvement in vision from moving bodies to 6/60.

*Radiation therapy.* The patient was then referred to Tata Memorial Cancer Institute in Bombay for irradiation with deep X rays. The dose suggested was 200 r, every day for six days, with Philips contact apparatus, at 55 kv. This irradiation was prescribed for secondary vascularization and for the graft area which actually covered the region where the tumor had been.

Six weeks after radiation therapy, vision improved to 6/18. There is now a thin corneal opacity on the medial side of the cornea. Now that the interior of the eye could be seen, it was observed that there were a few peripheral lens opacities and also sclerosis of the lens. The fundus was normal. The site of the original growth has remained clear and there is almost no evidence of the extensive grafting.

SUMMARY

Bowen's disease is discussed and some of the literature reviewed. A case report is presented. Treatment consisted of radical surgery followed by deep X-ray irradiation (contact). The results are highly satisfactory and gratifying. It is believed that, after radical excision of the tumor, the dosage of the irradiation needed is nearly one-fourth of that previously reported.

*Nawapura Street.*

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# FURTHER STUDIES ON THE INFLUENCE OF ACTH ON THE REACTIVITY OF THE TERMINAL VASCULAR BED IN THE EYE*

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In a recent study (1953)[1] conducted in this laboratory, it was found that intravenous infusion of ACTH often potentiated the reactivity of the peripheral vascular apparatus of cats to adrenaline, noradrenaline, acetylcholine and electric stimulation of the cervical sympathicus nerve. Zweifach et al.[1,2] who investigated the effect of ACTH and cortisone on the terminal vessels of the rat mesappendix, reported a similar increase in adrenaline responses. In the aforementioned experiments on cats, cortisone infusions did not effect a measurable change in the responses. Therefore, the possibility that the ACTH was contaminated with pressor substances had to be considered. The fact that the phenomenon of sensitization of the terminal vasculature became visible during, or shortly after the infusion, gave added weight to the suspicion that impurities in the ACTH preparation contributed to, or even caused the observed increases of the induced responses.

No information was available on the pressor substance content of the one commercial ACTH preparation tested. It seemed advisable, then, to extend the study to determine whether the increased responses to various stimuli were the result of specific action of ACTH or of its contaminants. For this purpose, the changes of blood and eye pressure were observed after the infusion of small amounts of pure pressor substances such as pituitrin and pitressin, and after the use of other ACTH preparations with a very low content of contaminating pressor substances.

## MATERIAL AND METHODS

The technique followed closely in all steps that described previously (1952, 1953).[3,4] Young, mature cats in chloralose anesthesia were prepared for artificial respiration and cannulation of the right femoral artery and left femoral vein. The cervical sympathetic nerve on one side was isolated for electric stimulation. The intraocular pressure of both

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eyes and the blood pressure of the iliac artery were determined manometrically and continuous records obtained in the usual way.

The pituitrin* and pitressin† preparations each contained 20 pressor units/cc. After the test substance was diluted, in most instances 400 times, with 0.9-percent solution of sodium chloride, 0.5 unit was incorporated by intravenous infusion over a period of four to 12 minutes. In a few experiments, the dose was increased to one or two units. Usually the administration of the compounds was repeated after a lapse of 25 minutes. The effect of pituitrin was studied on four animals, that of pitressin on six.

Three preparations of adrenocorticotropin were tested:

1. Purified corticotropin gel of Wilson in which a 16-percent gelatin medium serves as a vehicle; one cc. is reportedly equivalent to 13.3 units of corticotropin activity when injected intravenously (five experiments).

2. Corticotropin solution of Wilson; 40 USP units of activity per cc. (13 experiments)—it is estimated that the content of posterior pituitary pressor substances in this preparation is less than 0.0005 USP units per unit of activity.

3. Armour special ACTH; a porcine ACTH with an activity of 20 IU/mg. and a vasopressor content of 0.2 unit/mg. (six experiments).

Prior to and after the infusion of the posterior pituitary pressor substance or the ACTH preparation, comparison was made of the responses to threshold doses of adrenaline, noradrenaline, acetylcholine and to electric stimulation of the cervical sympathetic nerve.

## Results

### Effect of intravenous infusion of pituitrin

The infusion of 0.5 IU (three experiments) caused, in two animals, a transient blood pressure rise of five and 35 mm. Hg respectively. Eye pressure increases of 1.0 and 1.5 mm. Hg accompanied these blood pressure increments (Table 1). In three instances, one unit of the pressor substance was given; the blood pressure rose, in two experiments, 10 and 15 mm. Hg and the eye pressure rose one mm. Hg in one of these. The greatest effect, that is, a blood pressure rise of 70 mm. Hg and an eye pressure rise of 2.5 mm. Hg, occurred in the one experiment in which two units were infused. In all cases these pressure changes lasted three to six minutes.

After the use of pituitrin, the responses of the intraocular pressure to intravenous injection of 0.1 μg./kg. noradrenaline indicated an increase (0.5 mm. Hg) in one of four preparations, whereas there was no increase in the effect of the other stimuli. In general, with the lower doses of pituitrin, the blood pressure changes produced by noradrenaline exceeded by 5.0 to 15 mm. Hg. those observed before the treatment; adrenaline and acetylcholine responses were usually not augmented.

The eye pressure changes induced by electric stimulation of the cervical sympathetic chain were not altered, but it should be noted that at least 15 minutes elapsed between the infusion and the application of this stimulus.

### Effect of intravenous infusion of pitressin

The infusion of 0.5 IU of pitressin effected greater rises of the blood and eye pressure—10 to 45 mm. Hg and 1.0 to 5.0 mm. Hg respectively—than those observed in the pituitrin experiments and positive results were obtained in all five experiments in this group (table 1). The higher doses (one and two units) caused the blood pressure but not the eye pressure to rise to higher values, that is, 65 to 75 mm. Hg. The pressure increments persisted for about six minutes in two experiments, in the other preparations for only three minutes.

In two experiments the amplitudes of the

* Pituitrin (surgical)—Parke, Davis & Co.
† Pitressin—Parke, Davis & Co.

TABLE 1

EFFECT OF INTRAVENOUS INFUSION OF PITUITRIN AND PITRESSIN ON INTRAOCULAR AND BLOOD PRESSURE AND THE RESPONSES OF THESE FUNCTIONS TO PHARMACOLOGIC STIMULI

| Compound | Cat # | Dose IU/Animal | *Increases*—Intraocular Pressure | | *Increases*—Blood Pressure | |
|---|---|---|---|---|---|---|
| | | | Level (mm. Hg) | Responses—Pharm. Stimuli* (mm. Hg) | Level (mm. Hg) | Responses—Pharm. Stimuli* (mm. Hg) |
| Pituitrin | 245 | 1.0<br>2.0 | None<br>2.5 | None<br>None | None<br>70 | None<br>A 15, N 5 |
| | 251 | 0.5<br>0.5 | 1.5<br>None | None<br>None | 30<br>35 | N 15<br>N 10 |
| | 253 | 0.5<br>1.0 | None<br>None | N 0.5<br>None | None<br>15 | None<br>N 15 |
| | 254 | 0.5<br>1.0 | 1<br>1 | None<br>None | 5<br>10 | N 15<br>None |
| Pitressin | 245 | 1.0<br>2.0 | 5<br>4 | None<br>A 1.0, N 0.5 | 65<br>75 | None<br>A 15, N 5 |
| | 246 | 0.5<br>0.5 | 5<br>5 | A 1.5, ACh 0.5<br>None | 35<br>45 Irreg. | ?<br>None |
| | 247 | 0.5<br>0.5 | 2<br>1 | N 1<br>None | 25<br>15 | N 5<br>None |
| | 248 | 0.5<br>0.5 | 1.5<br>1.5 | None<br>N 1 | 10<br>15 | N 5<br>N 5 |
| | 249 | 0.5<br>0.5 | 1.5<br>1.0 | A 1.0, ACh 2.0<br>N 0.5 | 10<br>10 | A 5, ACh 5<br>A 10, N 5 |
| | 250 | 0.5<br>0.5 | 2<br>None | N 2, ACh 1.5<br>None | 25<br>5 | A 10, N 10<br>ACh 15 |

* A = adrenaline, N = noradrenaline, ACh = acetylcholine.

respiration and pulse waves of the eye pressure records were greater after the infusion; the responses to either adrenaline or noradrenaline were moderately increased in all five experiments of the low dose group, and to acetylcholine in three instances. The increments were generally of the order of 0.5 to 1.0 mm. Hg. The state of increased responsiveness to the various stimuli lasted about 20 minutes. The use of pitressin did not appear to alter the effects of electric stimulation.

EFFECT OF PURIFIED CORTICOTROPIN GEL OF WILSON

Marked potentiation (doubling) of the effect of noradrenaline, adrenaline and acetylcholine on the eye and blood pressure followed the infusion of three units of this preparation per kg. in only one of the five experiments. This animal responded to an infusion of ACTHAR in a similar fashion and to a similar degree. In one animal, the infusion of this preparation caused a transient marked fall of eye and blood pressure; such a reaction was never observed in any of the other experiments with ACTH in aqueous vehicles. Four experiments of this group were listed as negative with respect to sensitization to adrenergic and cholinergic stimuli, although in some of them moderate prolongation of these responses resulted from the ACTH infusion. There was no augmentation of the effect of electric stimuli.

EFFECT OF ARMOUR SPECIAL ACTH

In one of six experiments, elevations in

both the eye and blood pressure and increased responses to pharmacologic and electric stimulation followed the infusion of this preparation in doses of three and six units/kg. In another experiment, in which six units/kg. were given, the only change was an increased response to adrenaline observed in both pressure tracings.

### Effect of corticotropin solution of Wilson

In contrast to the two preceding series with ACTH, the results of the 13 experiments of this group compared favorably with the observations reported in the experiments with commercial ACTHAR.[1] After the infusion of three units of this compound per kg., the eye pressure rose in two experiments, 3.0 and 3.5 mm. Hg. The responses to adrenaline, noradrenaline and acetylcholine increased 1.0 to 2.0 mm. Hg in four experiments, one of which showed a greater effect of electric stimulation. Thus about one third of the preparations exhibited a generalized increase in responsiveness to pharmacologic stimuli.

In six instances of this series, the intraocular pressure increased 0.5 to 2.0 mm. Hg. On the other hand, the changes in reactivity to the pharmacologic compounds were moderate and were usually limited to adrenaline or noradrenaline. The response to electric stimulation was greater in three experiments. The three remaining experiments did not indicate any sensitizing effects of the ACTH preparation on vascular responses of the eye.

In 12 of the 13 experiments, a blood pressure rise of 5.0 to 35 mm. Hg followed the infusion of the soluble ACTH. As a rule, the changes effected in the eye and blood pressure by ACTH were of the same character but markedly disproportional. There was also a relative independence of the increases in the base pressures from the changes in the responsiveness to the pharmacologic and electric stimuli after the infusion of ACTH, for example, preparations No. 260, 263, 270, as shown in Table 2.

In most of the positive experiments, signs of sensitization to pharmacologic stimuli lasted around 30 minutes.

### Comment

In the previously reported extensive experimental series with ACTHAR, occasional rises of 5.0 mm. Hg occurred in the eye pressure. Such increases were not observed in any of the experiments described in this study, but a rise of 3.0 mm. Hg. occurred in several instances. After the treatment with the purified ACTH preparation of Wilson, which compound contains a minimal contamination with posterior pituitary pressor substances, one third of the experiments showed marked potentiation of the effects of pharmacological stimuli.

The effects were considered moderately positive in six experiments of this series and negative in three. This ratio approximates that obtained in the study with ACTHAR. The results of electric stimulaation were less consistent and did not always coincide with the positive responses to the pharmacologic agents. Direct comparison of this effect with the ACTHAR series is not possible because, in the latter case, this stimulus was tested in only 17 of 63 preparations.

At variance with the results obtained with Wilson soluble ACTH are the experiments with Wilson ACTH gel and Armour special ACTH in which usually no sensitization to the two types of stimuli could be demonstrated. Interference of the gel vehicle with rapid diffusion and proper distribution of the hormonal substances may account for the negative results with the corticotropin preparation of Wilson. No explanation can be advanced at the present time for the negative results with the new Armour preparation. The small amount of material available to us precluded additional investigations with this compound.

The similar results obtained with the Wilson soluble preparation clearly indicated that

TABLE 2

EFFECT OF INTRAVENOUS INFUSION OF WILSON CORTICOTROPIN SOLUTION ON INTRAOCULAR AND BLOOD PRESSURE AND THE RESPONSES OF THESE FUNCTIONS TO PHARMACOLOGIC AND ELECTRIC STIMULI

| Prep. No. | Dose (units/kg.) | *Increases*—Intraocular Pressure | | | *Increases*—Blood Pressure | | |
|---|---|---|---|---|---|---|---|
| | | Level (mm. Hg) | Responses | | Level (mm. Hg) | Responses | |
| | | | Pharm. Stimuli* (mm. Hg) | Electric Stimuli (mm. Hg) | | Pharm. Stimuli* (mm. Hg) | Electric Stimuli (mm. Hg) |
| 260 | 3<br>3 | 3<br>2 | None<br>None | None<br>None | 10<br>None | None<br>None | None<br>None |
| 261 | 3<br>6 | 3.5<br>3 | A 1.5, N 1, ACh 2<br>N 2 | None<br>None | 15<br>None | N 10, ACh 10<br>N 15 | None<br>None |
| 262 | 6<br>12 | 1<br>None | None<br>None | None<br>None | 15<br>None | None<br>None | None<br>None |
| 263 | 3<br>6 | 1<br>None | A 1, N 2<br>None | None<br>None | 10<br>10 | A 15, N 15, ACh 40<br>None | None<br>None |
| 264 | 6 | 1 | N 1 | Not Tested | 25 | A 10, N 15 | Not Tested |
| 265 | 3<br>6 | 1<br>1 | N 1<br>None | None<br>None | None<br>None | None<br>None | None<br>None |
| 266 | 3<br>6 | 3<br>3 | A 1.5, N 1, ACh 1<br>ACh 0.5 | 1.5<br>None | 30<br>25 | A 5, N 5, ACh 5<br>A 10, ACh 10 | None<br>None |
| 267 | 3<br>6 | None<br>None | A 0.5<br>None | None<br>None | None<br>10 | A 15, ACh 10<br>A 10 | None<br>None |
| 268 | 3<br>6 | 1<br>2 | None<br>N 0.5 | None<br>0.5 | 25<br>25 | A 15, ACh 10<br>A 5, N 5 | None<br>None |
| 269 | 3<br>6 | 2<br>None | A 1<br>None | 1.5<br>None | 35<br>5 | A 10, ACh 10<br>A 5 | None<br>None |
| 270 | 3<br>6 | None<br>1 | N 1, ACh 1<br>N 2, ACh 0.5 | None<br>None | None<br>10 | N 10, ACh 10<br>N 15, ACh 5 | None<br>None |
| 271 | 3<br>6 | 1<br>None | N 0.5<br>None | 1<br>None | 10<br>20 | N 15, ACh 10<br>N 10 | None<br>None |
| 272 | 3<br>6 | 0.5<br>None | None<br>None | None<br>None | 5<br>25 | None<br>None | None<br>None |

* A = adrenaline, N = noradrenaline, ACh = acetylcholine.

the sensitization of the terminal vascular bed which became evident after the infusion of ACTHAR could not have been influenced decisively by contamination of the corticotropic preparation with posterior pituitary pressor substances. The total amount of pressor substance introduced with the Wilson preparation did not exceed 0.00045 units per animal.

Infusion of isolated posterior pituitary pressor substances (pituitrin and pitressin) 100 times in excess of the quantity present in the pituitary preparation of Wilson caused, as expected,* a rise of the blood pressure accompanied by elevation of the intraocular pressure and, occasionally, by a moderate sensitization of the response to pharmacologic and electric stimuli. In contrast to the experiments with ACTH, the elevation of both pressure levels occurred frequently and lasted for a short period which did not exceed three to six minutes.

Hyperexcitability to pharmacologic stim-

* Duke-Elder demonstrated (1931[4]) the pressor effects of massive doses of pituitrin on eye and blood pressure of anesthetized dogs.

uli was a relatively rare and transient event. Infusion of Wilson soluble and Armour ACTHAR ACTH produced effects which persisted for 30 minutes to one hour and thus were quantitatively and qualitatively dissimilar from those obtained with infused pressor substances. Although the possibility cannot be excluded that contamination of the ACTHAR preparation contributed to the dramatic effects obtained previously, the present observations support the validity of the original interpretation, that is, ACTH alters characteristically the reactivity of the terminal vascular bed in cats under the experimental conditions described.

In view of the observations of Kurland and Freedberg[5] that striking potentiation of blood pressure responses to noradrenaline occurs in normotensive human subjects after treatment with cortisone, the results of these animal studies may be applicable to the human eye and explain certain early subjective and objective benefits encountered with the use of ACTH in clinical ophthalmology.

## Summary

An ACTH preparation with very low content of pituitary posterior-lobe pressor substances elicited changes of the intraocular pressure and blood pressure similar to those observed previously with an ACTH preparation of unknown contamination with these pressor substances. The ACTH-induced increase of the reactivity of the vascular terminal bed to adrenaline, noradrenaline and acetylcholine was similar in both series.

Infusion of pitressin and pituitrin in doses of 0.5 IU produced a short lasting rise of both pressures and occasionally, for a brief period, a small increase of the vascular responses to adrenaline, noradrenaline, and acetylcholine.

The positive responses which followed the injection of pitressin and pituitrin differed qualitatively as well as quantitatively from those produced by the ACTH preparations.

In addition, the amount of pure pressor substance required to effect such responses was about 100 times in excess of that injected as a contaminant with the ACTH preparation.

Therefore, the increase observed in the reactivity of the terminal vascular bed in the eye to adrenergic and cholinergic mediators after the infusion of two ACTH preparations is not attributable to contamination by these two pressor substances.

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# THE CLINICAL USE OF THE HAIDINGER'S BRUSHES PHENOMENON

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## Introduction

Haidinger's brushes is the name given to a phenomenon first described in 1844 by Wilhelm Karl von Haidinger which results when polarized light enters the eye. This can be readily seen by looking against the blue sky through a Nicol prism, a very efficient polarizer.

A summary of the important facts regarding polarization of light and the mechanism of production of Haidinger's brushes is of value here because most ophthalmologists have not concerned themselves with this subject. This will aid in the understanding of the basis for the clinical visualization of Haidinger's brushes.

Light transmission is a wave phenomenon with vibrations in all planes. Polarization of light occurs when all planes of vibration have been removed but one. This occurs naturally and can also be produced artificially in several ways.

Light may be passed through a doubly refracting substance. Such a substance is one in which the index of refraction is not the same in all planes. These substances are usually crystals and the property is dependent upon the crystal lattice structure. There is a definite axis in such a crystal.

For example, a Nicol prism is a crystal (Iceland spar) which has been sliced diagonally and the halves reunited with Canada balsam. The diagonal slice is at such an angle that incident light will be broken up into a transmitted component and an internally reflected component. The plane of vibration of the former will be in the axis whose index of refraction permits passage of light through the crystal. The plane of vibration of the latter will be in an axis with a different index of refraction and will strike the diagonal surface at such an angle as to be internally reflected. The transmitted component will then be polarized.

In nature, polarization of light is more often the result of reflection from reflecting surfaces particularly water. Water, of course, partially reflects light and partially refracts it. When light strikes an interface between air and water at such an angle that the axis of the refracted ray is exactly perpendicular to that of the reflected ray the latter is completely polarized. This angle may be called the critical angle. The exact reason for this has never been completely explained but has to do with electromagnetic vibrations set up in the reflecting substance by the incident light ray. Rays reflected at angles other than the critical angle are only partially polarized.

Light is normally more or less polarized by being passed through doubly refracting bodies, through dim media, and through the atmosphere due to reflection from water droplets. There is additional polarization of light in the eye itself.

The vitreous acts as a doubly refracting body, the major axis of which has an approximately horizontal direction with a slight inclination nasalward. The reflection of light within the eye and the scattering by heterogeneous ocular media produce further polarizing effects. These factors, of course, are variable.

When looking at the blue sky, Haidinger's brushes may be seen. They consist of a relatively dark yellow spindle-shaped figure, four to five degrees in angular size, flanked by bluish, hyperbolic regions. The axis of the hyperbolas corresponds to the plane of polarization. This phenomenon is the visual result of the absorption of the polarized component of sky light by the yellow doubly refracting lutein pigment in the macular area.[1] However, though theoretically possible, very few individuals can be made aware of Haidinger's brushes by merely looking at the sky because of incomplete polarization

and the variable factors already mentioned.

### Clinical use

In order to make use of this phenomenon for clinical testing of disturbances involving the absorption of polarized light by the lutein pigment, some device is required which will make it possible for all persons with normal eyes and average intelligence to see Haidinger's brushes readily under the conditions of the test.

Such an instrument is now available for clinical use.* Basically, it consists of a polarizing device for blue light at which the subject looks. The polarizer is made to rotate in order to make Haidinger's brushes more readily noticeable. The direction of apparent rotation can also be changed as a check on the accuracy of the subject's observations.

Evidence is available that the cornea, lens, and other ocular media have nothing to do with the perception of Haidinger's brushes, and that it is a function solely of the lutein pigment in the macular area.[2] This pigment is found in the outer plexiform layer of the retina which consists primarily of the axons of the first neuron whose dendrites are the cones, and the supporting fibers of Müller which run from the external limiting membrane to the internal limiting membrane passing through this layer. It will be recalled that because of the foveal pit the course of these axons is radially away from the fovea rather than toward the anterior surface of the retina as elsewhere.

In this parafoveal area the outer plexiform layer is referred to as the fiber layer of Henle. Therefore, any lesion which damages the cone cells, the axons of which contain lutein pigment, will prevent subjective visualization of Haidinger's brushes in polarized light. Of course, if the media are opaque enough or if the papillomacular bundle is badly enough damaged, recognition of Haidinger's brushes will also be interfered with.

* This instrument is obtainable from the American Measuring Instruments Corporation, 240 West 40th Street, New York. It is referred to as the Macula Deficiency Tester.

### Present study

The clinical usefulness of such recognition and the limitations placed thereon by opacities in the media and papillomacular bundle disturbances are the subjects of this study.

A series of ocular disturbances other than macular lesions was first studied to determine their effect upon the recognition of Haidinger's brushes. Age, sex, and race have no bearing on this. The youngest patient who could see the brushes in this series was five and one-half years of age, the oldest, 85 years. Several individuals of much less than average intelligence readily perceived the brushes.

The test is practically independent of the refractive error. Individuals with correctible visual acuity with errors ranging from −13.0D. sph. to +10.0D. sph. were able to recognize the phenomenon with or without glasses. Astigmatic errors in this series ranged from −5.0D. cyl. ax. to +4.5D. cyl. ax. without preventing perception of the brushes. Aphakic eyes containing or free from retained capsule or cortex were able to see the brushes with and without glasses.

It was found that "shatterproof" lenses reversed the apparent direction of rotation of the brushes produced by the polarizer in the instrument. This is due to the effect of cellophane in the "shatterproof" glass which reverses the plane of polarization by 90 degrees.[3]

A patient with plastic contact glasses was tested. Plastic does not produce a cellophane effect.

Polaroid is a substance commonly used in sun glasses and for other reasons to polarize light.† When viewing the blue light in this

† This substance is presently manufactured[4] by heating sheets of a clear, tough plastic, polyvinyl alcohol, a doubly refracting substance, and then stretching it mechanically. This apparently produces a parallel molecular structure. The stretched sheet of polyvinyl alcohol is then dipped in an aqueous solution of iodine. A chemical reaction between the iodine and polyvinyl alcohol molecules results which forms the finished polaroid.

instrument through polaroid, the light disappears and reappears as the polarizer in the machine rotates the axis of the polarized light into the axis perpendicular to the transmitting axis of the polaroid and out again.

Ten adult patients with anisometropia and amblyopia of one eye were tested. The amblyopic eye had the higher refractive error, spheric or cylindric, and the other eye had normal visual acuity. Some gave a history of childhood squint. Pinhole vision in the amblyopic eye varied from 6/12 to 6/30. All these patients saw Haidinger's brushes with the amblyopic eye though more dimly than with the normal eye.

Visual acuity of less than 6/60 due to suppression is apparently too poor to perceive the brushes. Visual acuity of 6/60 is about borderline. This would be additional evidence of the cortical rather than macular origin or suppression in anisometropia with or without squint.

It follows that amblyopia in an eye with 6/30 or better acuity which does not see Haidinger's brushes is probably due to a macular or papillomacular bundle lesion, if the media are clear. Theoretically, this test could be applied to determine which cases of monocular squint might be benefited by occlusion of the better eye. Practically, however, only a few children are reliable observers for this test at an age when occlusion will be of much value.

A series of patients with opacities in the media were tested. It was found that corneal opacities reducing the visual acuity as far as 6/30 were not incompatible with visualization of Haidinger's brushes. Below 6/60 they could not be seen. Similar findings were recorded in patients with incipient cataracts.

Several patients with vitreous opacities due to uveitis could recognize the brushes at visual acuities down to 6/22. Below this the test was questionable or negative. This could have indicated either too much vitreous opacity or macular damage or both.

The clinical significance of these findings is clear. If this test is an indication of macular function, it will reveal a functioning macula in the presence of opacities in the media which reduce the visual acuity to 6/22 or 6/30. In many instances such opacities prevent an adequate view of the macular area with the ophthalmoscope.

A number of patients with various types of glaucoma were tested. As long as the papillomacular bundle was not damaged, Haidinger's brushes were readily visualized. In general, when on the central field examination the 1/1,000 white isopter no longer included the fovea, the patient could not see the brushes.

Patients with nonglaucomatous lesions of the optic nerve fell into two categories; those with papillomacular bundle lesions and those with a peripheral form of optic atrophy.

If the papillomacular bundle was involved, recognition of the brushes depended on the severity of the disturbance and whether partial or complete restoration of function had taken place. If on the other hand the atrophy was peripheral, recognition was unimpaired.

A nine-year-old boy with a peculiar cyst down in the cup of his left optic nervehead on the temporal side had visual acuity of 6/30 and a central scotoma in this eye. The right eye was normal. He was unable to see Haidinger's brushes with his left eye but saw them readily with his right eye.

A 27-year-old woman had had acute retrobulbar neuritis of the left optic nerve with reduction of visual acuity to 6/60 14 months before the test. No definite cause was discovered. At the time of this test, her visual acuity had recovered to 6/9 and there was only a slight residual paracentral scotoma to 3/1,000 red. There was no scotoma to blue. She could see the brushes with each eye easily.

In examining patients with lesions of the macular area, both of retinal and choroidal origin, this test has its greatest value. Minimal amounts of damage to the cone cells interfere with perception of Haidinger's brushes.

Such damage may be of intraretinal origin; for example, edema, hemorrhage, exudation, cyst formation. It may also be

due to choroidal disturbance interfering with the nutrition of the cone cells across the lamina vitrea from the choriocapillaris.

It may likewise be seen in congenital lesions or hereditary degenerations of the macula. It is of interest to note here, however, that three individuals with the ordinary form of red-green color-blindness but with normal visual acuity readily saw Haidinger's brushes.

Intraretinal macular lesions will almost always prevent recognition of the brushes in their early stages even in the presence of 6/6 visual acuity. This failure of recognition corresponds more or less to the development of a central scotoma for blue test objects. This is particularly frequent in conditions lumped together under the term central angiospastic retinopathy. Included also in this category are such conditions as diabetic retinopathy, hypertensive retinopathy, traumatic edema of the macula, and occlusion of the central retinal vein or artery.

In a man with a cilioretinal artery in his left eye there was an occlusion of the central artery. He had a small triangular field between the disc and fovea and 6/12 visual acuity. Nevertheless, he could not see Haidinger's brushes presumably because of the considerable residual macular edema from interruption of the main arterial blood supply.

Subretinal or choroidal lesions disturbing the nutrition of the macular cone cells will also prevent visualization of Haidinger's brushes. These lesions include subretinal hemorrhage or exudation, marked macular colloidosis, and choroidal sclerosis with resulting senile macular degeneration.

## Comment

This test is quick and simple and adapted for office use. The patient is seated in a dark room as for slitlamp examination and asked to look at the blue-light background in the instrument which is produced by turning on the electric switch. Whenever a normal eye looks against the blue background, Haidinger's brushes appear as a grayish propeller-shaped rotating shadow. The farther away from the blue-light background one views the brushes, the larger they appear. They are always directly in the visual axis.

By throwing a cellophane diaphragm in between the light source and the patient, the direction of rotation can be reversed at will by the examiner to see whether the patient is really seeing Haidinger's brushes.

The test is of value in determining whether the macular area is normal when opacities in the media prevent an adequate ophthalmoscopic view as, for example, in moderately advanced cataract where the prognosis for vision after extraction is uncertain. It is also of aid in establishing the existence of macular deterioration in long-standing cases of keratitis or uveitis.

Minimal amounts of macular edema difficult to identify with an ophthalmoscope will usually prevent perception of the brushes. Similarly, early senile macular degeneration may be diagnosed before definite ophthalmoscopic signs occur.

Whether or not macular colloidosis is interfering with nutrition across the lamina vitrea can readily be determined. Where the disease process is likely to be bilateral the better eye may be tested in this fashion for the earliest evidence of the disease.

In contusions of the globe this is a quick test for "commotio retinae."

## Summary

1. Haidinger's brushes are the entoptic projection of Henle's fiber layer in polarized blue light. The center of this phenomenon is at the point of fixation.

2. The visualization of Haidinger's brushes is prevented in the earliest stages of the macular diseases described and therefore serves as a useful clinical test.

3. The limitations placed on the clinical value of the test by opacities in the media are described.

4. The effects of other ocular disturbances on the recognition of Haidinger's brushes are discussed.

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# RELATIVE FREQUENCY AND MASS TREATMENT OF TRACHOMA IN SOUTHERN IRAN*

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## INTRODUCTION

The conditions of poverty and promiscuity favorable to a high relative frequency of trachoma unfortunately exist to a marked degree in southern Iran. Sidky and Freyche[1] have reviewed the rather inadequate data available from this area. The report of Sadoughi[2] presents a more favorable picture of the situation in the province of Fars than our experience indicates to be the case.

Chams, as cited by Wibaut,[3] likewise gives figures for the Persian Gulf area that appear too low. (Wibaut's map erroneously places the cities mentioned by Chams in Fars, rather than in Khuzistan.) In the absence of legal requirements regarding the disease in Iran, satisfactory data will remain difficult to obtain.

It is hoped that the experiences reported here, collected during residence in Shiraz from August, 1952, to May, 1953, will help provide some information regarding conditions in the province of Fars, which has a population of about two million people and will, in addition, add to our knowledge of the efficacy of mass treatment.

* From the Department of Public Health and Preventive Medicine, School of Medicine, University of Washington, Seattle, and the Public Health Cooperative Organization, Shiraz, Iran. This study was conducted as part of the program of the Technical Co-operation for Iran (Point Four), in which the senior author participated under the contract between the United States Department of State and the University of Utah.

## RELATIVE FREQUENCY

### GENERAL

The program of the Health Section of the Shiraz Regional Team, working in Fars under the Technical Co-operation for Iran (Point Four) involved, among other services, the operation of four mobile health teams. In addition to other trained personnel, each of these teams included one or two graduates of a special "trachoma class." This class, instituted under the general direction of L. H. O. Stobbe, M.D., with supplementary specialized ophthalmic training by one of us (M. F.), provided a group of intelligent young people, both male and female, capable of recognizing trachoma and administering medication, including massage.

Reports submitted by the mobile teams entering rural villages north and south of Shiraz and southwest toward Bushire indicated a relative frequency of trachoma of close to 100 percent in these areas. In most rural areas in the province of Fars, there has been little possibility for treatment, and the relative frequency of trachoma agrees with MacCallan's generalization[4] that "in a trachomatous country all children are infected within the first two years of life among the mass of the population. By the evolution of the disease, many of these attain a natural cure."

In the city of Shiraz, where treatment is available and where personal contact is re-

duced for the wealthier classes, the relative frequency of trachoma was considerably lower than in the rural areas immediately surrounding the city. However, careful examination of residents of nearby villages, using a hand slitlamp, showed a high relative frequency, probably exceeding 90 percent, in the rural areas.

These observations, to which can be added the more detailed data tabulated below, would indicate the relative frequency of trachoma in the rural areas of the province of Fars to be over 90 percent. This is in agreement with the statement of Naficy, cited by Sidky and Freyche,[1] that the trachoma rate is extremely high in the coastal provinces of the Persian Gulf, above all in Khuzistan, which lies northwest of Fars. Our findings indicate that the situation in Fars is no better than in Khuzistan.

### Study group

In order to evaluate the relative effectiveness of the drugs chosen for trial, it was necessary to select five schools in which there was no record of previous treatment. These conditions were met in the three schools in Ghasrodasht and the schools in Dehkorreh and Saadi. The three villages named are all adjacent to Shiraz, the most distant being not over five miles from the center of the city. All pupils lived within a mile of the school attended.

Examinations of all children in each school were conducted with the aid of a hand slitlamp. An individual record card was prepared for each infected child, with an evaluation of his clinical condition according to the notation recommended by the Expert Committee on Trachoma of the World Health Organization.[5] For simplification, the data recorded herein are in terms of the basic classification proposed by MacCallan.[6]

Tables 1 and 2 provide information on the pupils and the relative frequency of infection, as determined during February and March, 1953. The data presented indicate the incidence of infection was extremely high, with the majority of the cases falling in MacCallan's stage III. An ophthalmologist of the World Health Organization, who visited Shiraz in May, 1953, examined all the patients in each of the schools in our group, and confirmed the clinical classifications with almost complete correlation.

## Mass treatment

### Experimental subjects

The 616 infected pupils of the five schools named in Table 2 were considered an ideal group for the evaluation of the proposed treatment methods. The pupils were from low-income families, generally living within the village under the conditions of crowding, poor housing and unsanitary environment which are unfortunately characteristic of much of the area. In addition, the dust storms of late summer, the hot, dry summer climate, and the numerous flies undoubtedly were contributing factors, insofar as these can be accepted as involved in the spread of trachoma.

### Materials and Methods

Table 3 presents data on the drugs selected for each school. The sodium sulamyd

TABLE 1

Location of schools and enrollment

| Village | School | Enrollment | | Total | Mean Age (in years) |
|---|---|---|---|---|---|
| | | Boys | Girls | | |
| Dehkorreh | Mardaviz | 100 | 0 | 100 | 9.9 |
| Saadi | Boustan Saadi | 99 | 18 | 117 | 9.1 |
| Ghasrodasht | Maktabi | 143 | 0 | 143 | 11.5 |
| Ghasrodasht | Onsori | 208 | 0 | 208 | 9.9 |
| Ghasrodasht | Davari | 0 | 157 | 157 | 10.3 |
| Totals | | 550 | 175 | 725 | 10.2 |

TABLE 2

FREQUENCY OF TRACHOMATOUS INFECTION, BY STAGES, IN PUPILS OF FIVE SCHOOLS

| School | Number of Infected Pupils, (by stages) | | | | Number Infected | Total Pupils | Percent Infected |
|---|---|---|---|---|---|---|---|
| | I | II | III | IV | | | |
| Mardaviz | 12 | 25 | 39 | 6 | 82 | 100 | 82 |
| Boustan Saadi | 21 | 36 | 47 | 4 | 108 | 117 | 92 |
| Maktabi | 11 | 19 | 82 | 11 | 123 | 143 | 86 |
| Onsori | 24 | 44 | 94 | 9 | 171 | 208 | 82 |
| Davari | 24 | 44 | 62 | 2 | 132 | 157 | 84 |
| TOTALS | 92 | 168 | 324 | 32 | 616 | 725 | 85 |

(Schering), aureomycin (Lederle), and terramycin (Pfizer), both ointments and crystalline material for solutions, were purchased on the Tehran and Shiraz open markets in original unopened packages. Elkosin (Ciba) was provided for clinical trial through the kindness of the manufacturer. Copper sulfate and methylene blue solutions (this type of medication is locally favored) were compounded in Shiraz as follows:

*Copper-sulfate solution* (10 percent):

| | |
|---|---|
| Copper sulfate | 1.0 gm. |
| Bichloride of mercury | 0.10 gm. |
| Glycerol | 10.0 gm. |

*Methylene-blue solution* (0.1 percent):

| | |
|---|---|
| Methylene blue | 0.01 gm. |
| Novocaine | 0.10 gm. |
| Dist. water | 10.0 gm. |

It is regretted that a sixth school could not be selected to serve as an untreated control group, but personnel and transportation problems prevented such additional work. Furthermore, it was felt that the chronicity of the disease would insure a relatively static situation in any control group during the limited period of the experimental treatment. The contemplated one year follow-up study will, it is anticipated, include further surveys of untreated groups as controls.

The treatment program provided for the assignment of two of the graduates of the training program referred to above to each of the five selected schools. Each team was provided with lid retractors, applicators, cotton, disinfectant, and a quantity of the drug to be used sufficient for several days' treatment.

Antibiotic solutions were freshly prepared from the dried powder as needed, and were not used when potency was questionable. New supplies were issued as required, and

TABLE 3

LOCATION OF PATIENTS AND METHOD OF TREATMENT

| School | No. of Patients | Drugs Used | | Strength (percent) |
|---|---|---|---|---|
| Mardaviz | 82 | Elkosin | Solution<br>Ointment | 5.0<br>5.0 |
| Boustan Saadi | 108 | Sodium sulamyd | Solution<br>Ointment | 30.0<br>10.0 |
| Maktabi | 123 | Copper sulfate<br>Methylene blue | | 10.0<br>0.1 |
| Onsori | 171 | Terramycin | Solution<br>Ointment | 0.5<br>0.5 |
| Davari | 132 | Aureomycin | Solution<br>Ointment | 0.5<br>1.0 |

close supervision was exercised throughout the treatment period. Each school was visited by us at least once daily, and constant efforts were made to impress on the young people administering the drugs the need for careful and conscientious work.

The pupils of the four schools where both solution and ointment were administered received the drops twice daily, the first treatment being given between 8 and 9 a.m. and the second between 11 a.m. and noon. The third and final treatment of the day, between 3 p.m. and 4 p.m., consisted of moderate massage of the everted conjunctiva with the appropriate ointment, using cotton-tipped applicators.

In the fifth school, treatment was at the hours, listed above, and consisted of administration of methylene-blue solution twice in the morning. Copper-sulfate massage was administered every second afternoon, with methylene-blue drops being substituted on the intervening days.

Treatment as described above commenced on the morning of March 6, 1953, and continued until the end of the school day on June 6, 1953. During this period of 93 days, there were 25 days during which no treatments were given. The medication therefore was administered in each case for 68 days during a total elapsed period of 93 days.

## Results

Commencing about 40 days after the beginning of the trials, careful examination of each patient was again carried out, using a hand slitlamp. Since these re-examinations were spread over a period of about 10 days, it may be assumed for all practical purposes that half the treatment was completed at the time the first evaluation was made. Because of parental objections, withdrawals, absence from school and other reasons, all the patients were not available for study. Table 4 summarizes the observations at the midpoint of the study.

It was our opinion that the results summarized in Table 4 reflected to some extent the effect of the treatment on secondary bacterial infection. It was extremely regrettable that all efforts to obtain the supplies and equipment needed to carry out concurrent bacteriologic studies were futile.

The treatment schedule was continued during and after the midpoint examination. Following the completion of 68 days of treatment in each school, final examination was made of each patient.

Table 5 reports the results of the third examination of each patient. There was the expected attrition due to the factors noted above. Although in some cases there were absences from school during the treatment period, it may be assumed for all practical purposes that all those pupils evaluated in Table 5 had completed the full course of treatment, or had missed not more than four of the 68 treatment days.

Inspection of Table 5 reveals a rather marked improvement in some cases from the midpoint examination (table 4). Since the effect of the treatment on the concomitant bacteria had probably reached its maximum before or by the time of the midpoint examination, it seems reasonable to attribute the improvement of the second half of the treatment period to the effect on the primary disease itself.

It is of interest to speculate on the possible effects of a continuation of treatment beyond the 68-day period used in this trial. However, it must be pointed out that this study was undertaken in an effort to find a practical, financially feasible means for treatment of trachoma. Extension of treatment beyond the total 93-day elapsed period of this study seemed both impracticable and unlikely to provide useful information.

Consideration of the effect of treatment on different stages of the disease was of interest from the standpoint of further trials. Table 6 shows the breakdown by stages, together with the improvement on final evaluation. In calculating the percentage of cases still showing active trachoma after treatment, the stage IV figures were not included, since the disease process in these patients was inactive by definition.

TABLE 4

EFFECT OF TREATMENT AT MIDPOINT

| School | Drug | Original Patients | Number Re-examined | Clinical Evaluation | | | | | Clearly Improved (percent) |
|---|---|---|---|---|---|---|---|---|---|
| | | | | − | 0 | ± | + | ++ | |
| Mardaviz | Elkosin | 82 | 71 | 10 | 28 | 12 | 20 | 1 | 30 |
| Boustan Saadi | Sodium sulamyd | 108 | 86 | 8 | 40 | 20 | 17 | 1 | 21 |
| Maktabi | Copper sulfate | 123 | 112 | 11 | 54 | 17 | 26 | 4 | 27 |
| Onsori | Terramycin | 171 | 160 | 12 | 71 | 32 | 37 | 8 | 22 |
| Davari | Aureomycin | 132 | 113 | 3 | 27 | 32 | 41 | 10 | 45 |

− Clinical regression.
0 No change.
± Questionable improvement.
+ Definite improvement.
++ Marked improvement.

While some of the figures in Table 6 are too small to be of a marked degree of significance, they seemed to indicate, as might be expected, that treatment during the early stages of the infection offers the greatest possibility of reducing the number of active cases. However, attention must be directed to the fact that although there was some clinical improvement in most patients, the reduction in number of active cases was somewhat disappointing.

If we consider the difficulty in determining whether or not a "cure" has actually occurred, we are faced with the realization that the results reported herein indicate that with three daily treatments in eight hours we do not have available a satisfactory means for mass treatment of trachoma.

Table 6 would imply that the copper-sulfate method produced a greater proportion of apparent cures than the antibiotic treatments, and that the sulfa compounds were relatively ineffective in reducing the number of active cases when applied topically, although a significant number of cases were improved. However, the well-known undesirable effects of copper sulfate would preclude the use of this substance without the close supervision of a trained ophthalmologist. This is an obvious barrier to widespread use of the material in underdeveloped areas, where administration of treatment must necessarily be performed by subprofessional personnel specially trained for this work.

## DISCUSSION

A drug suitable for use in a program for mass treatment of trachoma in underdevel-

TABLE 5

EFFECT OF 68 TREATMENT DAYS: EVALUATION BY METHOD OF TREATMENT

| School | Drug | Original Patients | Number Completing Treatment | Clinical Evaluation | | | | | Clearly Improved (percent) | Improvement after Midpoint |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | − | 0 | ± | + | ++ | | |
| Mardaviz | Elkosin | 82 | 72 | 3 | 33 | 16 | 14 | 6 | 28 | Not Significant |
| Boustan Saadi | Sodium sulamyd | 108 | 99 | 2 | 24 | 27 | 36 | 10 | 46 | Moderate |
| Maktabi | Copper sulfate | 123 | 114 | 0 | 5 | 24 | 49 | 36 | 75 | Marked |
| Onsori | Terramycin | 171 | 143 | 0 | 11 | 46 | 67 | 19 | 60 | Marked |
| Davari | Aureomycin | 132 | 117 | 0 | 17 | 33 | 56 | 11 | 57 | Slight |

− Clinical regression.
0 No change.
± Questionable improvement.
+ Definite improvement.
++ Marked improvement.

TABLE 6

EFFECT OF 68 DAYS OF VARIOUS TREATMENTS AS RELATED TO STAGES OF TRACHOMA

| School | Drug | Status at Start | Number Completing Treatment | Final Clinical Evaluation | | | | | Clearly Improved* (percent) | Number with Active Trachoma | Cases Still Active† (percent) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | − | 0 | ± | + | ++ | | | |
| Mardaviz | Elkosin Drops and Ointment | I | 11 | 1 | 6 | 2 | 2 | 0 | 19 | 9 | 80 |
| | | II | 22 | 1 | 16 | 2 | 1 | 2 | 14 | 21 | |
| | | III | 36 | 1 | 10 | 12 | 9 | 4 | 36 | 25 | |
| | | IV | 3 | 0 | 1 | 0 | 2 | 0 | — | 0 | |
| Boustan Saadi | Sodium sulamyd Drops and Ointment | I | 20 | 2 | 10 | 4 | 4 | 0 | 20 | 17 | 70 |
| | | II | 31 | 0 | 7 | 10 | 8 | 6 | 51 | 25 | |
| | | III | 45 | 0 | 7 | 11 | 23 | 4 | 60 | 25 | |
| | | IV | 3 | 0 | 0 | 2 | 1 | 0 | — | 0 | |
| Maktabi | Copper sulfate and Methylene blue | I | 10 | 0 | 2 | 1 | 7 | 0 | 70 | 2 | 14 |
| | | II | 17 | 0 | 0 | 2 | 9 | 6 | 88 | 6 | |
| | | III | 76 | 0 | 3 | 18 | 26 | 29 | 72 | 6 | |
| | | IV | 11 | 0 | 0 | 3 | 7 | 1 | 73 | 0 | |
| Onsori | Terramycin Drops and Ointment | I | 20 | 0 | 0 | 7 | 13 | 0 | 65 | 3 | 48 |
| | | II | 33 | 0 | 5 | 7 | 13 | 8 | 64 | 27 | |
| | | III | 82 | 0 | 6 | 28 | 37 | 11 | 59 | 35 | |
| | | IV | 8 | 0 | 0 | 4 | 4 | 0 | — | 0 | |
| Davari | Aureomycin Drops and Ointment | I | 21 | 0 | 0 | 6 | 15 | 0 | 71 | 0 | 29 |
| | | II | 41 | 0 | 7 | 9 | 20 | 5 | 61 | 20 | |
| | | III | 53 | 0 | 10 | 18 | 19 | 6 | 47 | 13 | |
| | | IV | 2 | 0 | 0 | 0 | 2 | 0 | — | 0 | |

* Indicates percentage of patients in each stage who showed definite improvement. Stage IV cases not significantly numerous.

† Figures in this column do not include stage IV cases, inactive at start.

− Clinical regression.
0 No change.
± Questionable improvement.
+ Definite improvement.
++ Marked improvement.

oped areas should ideally have the following characteristics:

1. It should be nontoxic to humans.
2. It should cure or arrest the disease in a high percentage of cases.
3. It should be stable without refrigeration under tropical weather conditions.
4. It should be readily available and inexpensive.
5. It should be capable of administration by subprofessional personnel after a minimum of training.
6. It should not require a prolonged period of administration.

Unfortunately, these ideal properties do not exist in any drug, or combination of drugs, presently available.

It is generally agreed that the eventual control of trachoma will be relatively simple when the environmental sanitation of the endemic areas is sufficiently raised. This, of course, must be accompanied by an improved educational pattern. From a realistic standpoint, however, we must face the fact that control through these measures in many parts of the world is years in the future. In the meantime, it seems only reasonable to attempt to reduce the size of the reservoir of infection by all available means.

The recommendations of the Expert Committee on Trachoma of the World Health Organization[6] for mass treatment of uncomplicated cases call for application of aureomycin or terramycin (one-percent ointment four times daily) without interruption for a period of two months.

On this basis, our medication was inadequate. However, we wish to point out that in

areas where reliable personnel are few in number, and where supervision of the administration of each treatment in each individual is essential, the recommendations of the Expert Committee for uninterrupted treatment at regular intervals are somewhat unrealistic.

There seems to be general agreement that antibiotic therapy provides a satisfactory means for the treatment of trachoma *provided* the drug is administered with sufficient frequency to maintain a virustatic concentration at all times during the treatment period. Mass treatment programs which do not, or cannot, provide for such medication will not yield a satisfactory number of cured or arrested cases. Our program, which was based on the maximum possible number of treatments during the school day, apparently did not result in maintenance of an adequate drug concentration.

Thygeson[7] has pointed out that trachoma therapy is no longer a problem under conditions of private practice, where regular administration of antibiotic ointment can be relied on to maintain an adequate concentration of the drug throughout the entire treatment period. It seems apparent that mass treatment of trachoma will be effective only so long as the recognized principles of antibiotic therapy are accepted and adhered to. Treatment schedules which can not meet the requirement for maintenance of virustatic concentrations of antibiotics will therefore fall short of adequate results. There can be no successful compromise with this requirement for adequate treatment with our presently available drugs, in spite of the extreme difficulty of such a program from the practical standpoint.

Another possible point of disagreement with the Expert Committee is in the method of administration of ointments. The recommendations calling for the use of the antibiotic ointments without massage have the advantage of simplicity but, in our opinion, with a possible reduction in effectiveness.

In numerous cases in which follicles in the conjunctiva of the upper lids disappeared after a few weeks of the treatment already described, many follicles could still be found in the conjunctiva of the lower lids, and in the plica semilunaris. Our results indicate that the follicles will not regress markedly without massage.

As Thygeson points out,[7] however, there is a definite lag between disappearance of the virus and disappearance of the follicles. Since our project was terminated after final examination of the patients, we are unable to state whether absorption of the follicles continued following cessation of treatment. In view of the likelihood that our treatment schedule was inadequate, as discussed above, it would be reasonable to predict that persistence of some follicles could be expected. A re-examination of the subjects may resolve this question.

In view of the cost of antibiotics, particularly where solutions are to be used, complete reliance on these materials can hardly be considered where large groups of victims of the disease are involved. There is obviously an urgent need for the development of more suitable substances, particularly if one considers the high incidence of the disease in underdeveloped areas, where marked improvement in general standards of sanitation and education lies years in the future.

The results reported here do not point out a satisfactory answer to the unsolved problem of adequate means for treatment. The unsupervised use of caustic solutions is out of the question. Under practical conditions in endemic areas, particularly where these areas are distant from modern transportation, it can hardly be expected that the recommendations of the Expert Committee for uninterrupted administration of antibiotic ointments can be met except for populations under closely supervised control, such as in refugee camps.

Our findings indicate that aureomycin and terramycin do not give adequate numbers of arrested cases under the dosage and treatment schedules we were able to follow. Pos-

sibly a combination of massage with antibiotic ointment and administration of methylene-blue solution might give improved results without the objectionable feature of high cost and unstability of the antibiotic solutions.

There is reason to feel, as already discussed, that our treatment schedule was inadequate and that more frequent administration of medication, particularly with the antibiotic ointments, would have yielded an entirely different set of results. Nevertheless, it is our opinion that the schedules followed represented the maximum program which could reasonably be planned with due consideration of available personnel and finances, and in view of the need for minimal interruption of the daily routine.

While it must unfortunately be assumed that relapses and reinfections will occur in an unknown proportion of cases treated on a mass basis, nevertheless a defeatist attitude is not justifiable. Continuous efforts must inevitably reduce the sources of infection. The extent of reduction will depend on the thoroughness and vigor with which mass treatment is introduced, and the degree to which it can be expanded from the starting point.

The cultivation of the virus of trachoma in the laboratory would certainly lead to a rapid advance in knowledge of many of the unsolved problems of the disease and its causative agent. There would probably be no more fruitful approach to eventual control of the disease than expansion of efforts in this direction.

## Summary

1. Field reports from the province of Fars, in southern Iran, indicate a relative frequency of trachoma of over 90 percent in rural areas.

2. In five schools on the outskirts of Shiraz, where slitlamp examination showed a relative frequency of trachoma of 85 percent, thrice-daily treatment was instituted for 616 children. Local applications of Elkosin, sodium sulamyd, copper sulfate-methylene blue, terramycin, and aureomycin were each used in one school.

3. Final evaluation after 68 days of treatment in an elapsed period of 93 days showed, in 545 children completing the schedule, that improvement was most noticeable in the group receiving the copper sulfate-methylene blue treatment. In order of decreasingly noticeable improvement were terramycin, aureomycin, sodium sulamyd, and Elkosin.

4. The percentage of cases still judged as clinically active following treatment was: For copper sulfate, 14 percent; aureomycin, 29 percent; terramycin, 48 percent; sodium sulamyd, 70 percent; and Elkosin, 80 percent.

5. The failure to obtain adequate numbers of arrested cases can be at least partially explained on the grounds of inability to maintain virustatic concentrations of the selected medication under the conditions of the treatment program.

6. The urgent need for the development of more adequate means of treatment, particularly with a realistic view of the problems of mass medication in underdeveloped areas, is discussed.

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## THE CILIARY BODY IN RADIATION CATARACT*

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The study of the etiology of cataracts has generally been along two lines: (1) the study of the supporting and nutritive structures, such as the aqueous and the ciliary body, and (2) the study of the lens itself.

For many years the investigators have concerned themselves with the increase or decrease of the various constituents of the lens, and it has only been in the past several years that investigators have agreed that most of the changes measured are indicative of a dead or dying tissue and are not of etiologic significance.

Largely through the efforts of Friedenwald[1] and Kinsey,[2] the emphasis has now shifted to the surrounding structures of the lens, primarily the aqueous, ciliary body, subcapsular epithelium, and vitreous. This has been as true of radiation cataract as of any other type, and von Sallmann,[3] in 1951, gave an excellent historic survey of the work in this field.

With the reporting of cataracts after exposure to the atom bomb[4-6] and through the stimulus of the interest of the Atomic Energy Commission, the study of radiation cataracts received considerable impetus.

The morphologic changes in the lens following X-irradiation and exposure to fast neutrons have been reported in detail by Cogan and his co-workers.[7,8] Von Sallmann[3, 9] has carefully studied the effects of radiation on the mitotic activity of the lens epithelium and has discussed the protective effect of SH groups on this process.

From the foregoing work it may safely be concluded that in the lens itself the initial site of injury is the lens epithelium. However, it appeared possible that this injury might not be due to direct hits on the epithelium, but might be secondary to destruction of the nutritive function of the ciliary body. This seemed particularly appropriate since it could be postulated that the protective action of SH groups reported by von Sallmann might in reality be on the oxidation-reduction mechanism of the ciliary processes.[1]

Apparently a similar hypothesis was entertained by Alter and Leinfelder,[10] since in a recent paper they gave the results of a series of experiments similar to those to be reported here that rather conclusively implicate the lens epithelium as the site of the initial lesion in roentgen-ray cataract.

Our interest in the role of the ciliary body as one of the offenders in the development of radiation cataracts arose from reports of altered permeability of this structure to fluorescein, sodium, and potassium[11] after irradiation, and from our own preliminary experiments using radioactive iodoalbumin as an indicator. To assay the relative importance of this altered permeability we undertook a series of experiments which we hoped would selectively injure either the lens or ciliary body, but not both. The results of this investigation are here reported.

* From the Department of Ophthalmology, Northwestern University Medical School. This report is part of a study on radiation cataract performed under contract #AT (11-1)-114 of the Atomic Energy Commission. The major portion of this paper was presented at the 89th annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1953.

## Experimental Techniques

The experimental animal in all these studies was the chinchilla rabbit weighing three kilograms ±0.2 kg. In every case the right eye was irradiated, the left eye being used as a control. The total dose of X rays was a single exposure of 4,000 r. Although this is a higher dose than is needed to produce opacities in the lens, it was found that with such a level of X rays the cataracts produced followed a more uniform course and in most cases went on to form dense mature cataracts, which was not true when smaller doses were used. The radiation factors were as follows: 220 kv., 20 ma., 122 r per minute in air, 0.5 mm. Cu. and 1.0 mm. Al filtration, 30 cm. target-skin distance, 20 mm. field (round), half-value layer 1.05 mm. Cu.

The rabbits were anesthetized with sodium pentobarbital, placed on their left side, the lids taped back, and the right eye irradiated via a lead shield which protected the drawn-back lids.

In an attempt to radiate selectively different portions of the eye, lead shields were prepared with different types of apertures. The shields were made by taking a plaster-cast impression of the eye with the lids retracted, preparing a brass die, and stamping out lead shields which were then perforated as desired. These lead "contact lenses" stayed in position during radiation as long as anesthesia was adequate. The rabbit was constantly observed during the irradiation period and at the slightest movement the X-ray machine was turned off and the position of the shield inspected.

These shields were of three types:

*Type I* had a central aperture and two sizes were made, with 5.0-mm. and 6.5-mm. circular openings.

*Type II* had a peripheral aperture 1.0-mm. wide, and again there were two subtypes; one had a 10-mm. aperture with a lead disc protecting the central 8.0 mm. of lens, and the second had a 12-mm. aperture with a central lead disc 10-mm. in diameter.

These sizes were deliberately chosen, since in a series of measurements it was found that the lens in a 3.0-kg. rabbit varied from 9.5 to 10 mm. in diameter and the secretory portion of the ciliary body was 1.5 mm. in width.

It was thus hoped that with the Type I shield it would be possible to irradiate the periphery of the lens only, and with the second type to irradiate the ciliary body alone.

*The Type III* shield had a semicircular aperture with a radius of 6.5 mm. through which one half of the whole lens and ciliary body could be irradiated.

Figure 1 is a photograph of X-ray film exposed to a short burst of X rays via the lead shields described above. The blackening of the film corresponds to the aperture in the lead shields. A is the shield with the central aperture, B is the shield with the peripheral aperture, and C is the shield with the semicircular aperture. Note the sharpness of the outline of the exposed film, indicating very little spread of the X rays.

Following the publication of the paper of Alter and Leinfelder,[10] it was felt that per-


Fig. 1 (Puntenney and Shoch). X-ray pattern of shield apertures. (A) Central aperture. (B) Peripheral aperture. (C) Semicircular aperture.

haps more exact delimitation of the beam of X ray might be obtained by suturing the lead "contact lens" to the eye and in the next two series of rabbits this was done. In one series of six rabbits a lead "contact lens" with a 10-mm. central aperture was sutured to the sclera and in the second series a 10-mm. disc was imbedded in a plastic contact lens and the latter was sutured to the sclera so that the lead disc overlay the lens.

The rabbits were examined at weekly intervals after irradiation and notes were made of the time of onset of opacities, vacuoles, and so forth. Their progression was watched and the time of complete opacification recorded. At varying intervals of time the animals were killed, the eyes removed and opened by halving the globe in a frontal plane. Gross changes were photographed and the anterior segments imbedded for sectioning and microscopic study. It was found that by cutting the section parallel to the plane of the iris a goodly portion of the ciliary body could be seen on one section.

## Results

### Measurements of lens and ciliary body

Figure 2 shows a normal eye removed from a 3.0-kg. rabbit and dissected by removing the posterior segment. This is a view into the eye from behind, maintaining the normal curvature of the anterior segment. A transparent ruler was placed over the eye. Note that the lens is 9.5 to 10-mm. in diameter and the secretory portion of the ciliary body is about 1.5 mm. in width. It is particularly interesting to see the close relationship of the ciliary body to the lens. The anatomic situation made us a little skeptical of the feasibility of irradiating one structure without the other, but this will be elaborated on later.

### Selective radiation

*No shield*

In Table 1, the Group I line shows the results in a series of 20 rabbits without any lead shielding. The opacities developed in 100 percent of the animals in six to seven weeks, and of this group 18, or 90 percent went on to complete opacification in three to five months.

*Central aperture* (5.0, 6.5, and 10 mm.)

In Table 1, the Group II material shows the results in a series of 13 rabbits irradiated via a lead shield with a central aperture.

Of the seven rabbits for which a 5.0-mm. aperture was used, one developed a minute subcapsular cluster of vacuoles, and after 11 months the remaining six rabbits still have absolutely clear lenses.

In the group of six rabbits, for which a 6.5-mm. aperture was used, a similar cluster

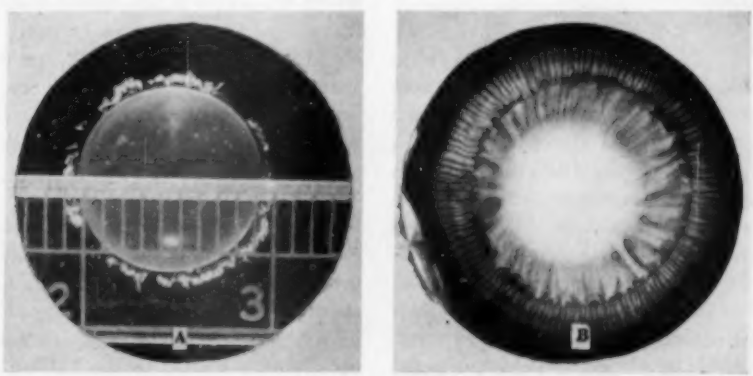


Fig. 2 (Puntenney and Shoch). Measurements of lens and ciliary body in normal three-kg. rabbit. (A) Lens. (B) Lens and ciliary body.

TABLE 1

DATA ON STUDY OF RADIATION CATARACTS

| Group | Shield | No. of Rabbits* | No. with Cataracts | Onset (weeks) | No. of Mature Cataracts | Time of Maturity | Comment |
|---|---|---|---|---|---|---|---|
| I | None | 20 | 20 | 6–7 | 18 | 3–5 months | — |
| II | Central aperture | | | | | | |
| | A: 5 mm. | 7 | 1 | — | 0 | — | At 11 mo. 6 lenses still clear |
| | B: 6.5 mm. | 6 | 1 | — | 0 | — | At 7 mo. 5 lenses still clear |
| | C: 10 mm. (sutured to globe) | 6 | 6 | 6–7 | 0 | — | At 9 mo. |
| III | Peripheral aperture | | | | | | |
| | A: Central disc 8 mm. | 6 | 6 | 6–7 | 0 | — | At 10 mo. |
| | B: Central disc 10 mm. | 6 | 1 | — | 0 | — | At 5 mo. |
| | C: Central disc 10 mm. in plastic sutured to globe | 5 | 4 | 6–7 | 0 | — | At 9 mo. |
| IV | Semicircular aperture | 6 | 5 | 6–7 | 0 | — | At 5 mo. all opacities limited to area irradiated |

* All rabbits received 4,000 r to right eye.

of vacuoles developed in one rabbit, and after seven months the remaining five rabbits showed no opacities of any type. The total incidence was two in 13, or 15 percent.

In the third series of rabbits in this group in which a 10-mm. central aperture was used opacities developed in 100 percent of the animals in six to seven weeks. These opacities were equatorial and/or posterior subcapsular and migrated anteriorly and posteriorly in the cortex. However, none became mature.

*Peripheral aperture*

In Table 1, the Group III material gives the results obtained in a group of 17 rabbits exposed to 4,000 r via a lead shield with a peripheral aperture. In this group there is an interesting and important difference seen with the use of the two different sized apertures.

1. *A 10-mm. aperture with an 8.0-mm. central lead disc.* In this series of six rabbits where the equator of the lens was exposed, all six animals developed vacuoles and granules in six to seven weeks, but at the end of 10 months none of these lenses had progressed beyond the incipient stage.

2. *A 12-mm. aperture with a 10-mm. central lead disc.* In this series of six rabbits where the secretory portion of the ciliary body was exposed, one rabbit developed a minute vacuole cluster which remained unchanged at the end of five months. The other five lenses have remained clear.

3. *A 10-mm. central lead disc* exposing the rest of the eye. In this series of five rabbits where the lens was supposedly shielded, but the whole rest of the eye exposed, four of the five rabbits developed granules and vacuoles after six to seven weeks none of which progressed to maturity in a period of nine months.

*Semicircular aperture*

In Table 1, the Group IV material shows the results in the group where only half of the eye was exposed. In this group five of the six rabbits developed clusters of vacuoles in six to seven weeks, and in all cases the opacity was limited to the half of the lens exposed. At the end of five months none of

these has gone on to complete opacification, and the sixth animal, a rugged individualist, still shows no opacities.

## Anatomic studies

### *Gross*

The appearance of these eyes after irradiation is illustrated by Figures 3 and 4. Figure 3 is a view from the back of a normal eye. Figure 4-A shows a similar preparation 48 hours after irradiation. Note the many hemorrhages in the ciliary body. Figure 4-B is a similar preparation 48 hours after irradiation via the peripheral aperture exposing only the ciliary body; again gross hemorrhages are visible.

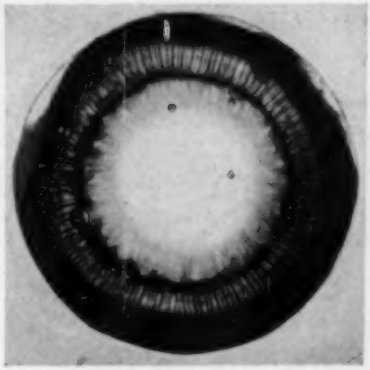

Fig. 3 (Puntenney and Shoch). Posterior view of normal lens and ciliary body.

### *Microscopic*

1. *Normal.* Figure 5 shows ciliary processes in a normal eye. This eye was sectioned parallel to the plane of the iris. The distinctness of the epithelial cells, the fine structure of the stroma, and the isolation of the individual processes are easily seen.

2. *Acute (48 hours after irradiation).* Figure 6 is a preparation similar to the one described above, photographed 48 hours after irradiation. The most prominent findings at this stage are hemorrhages, edema, leukocytic infiltration, and necrosis and vacuolization of the epithelium.

3. *Chronic (mature cataract).* Figure 7 is a section of the ciliary body five months after irradiation. Except for slight atrophy and vacuolization of the epithelium the structures appear almost normal. With the stain used here no changes in the vessel wall can be observed.

## Discussion

These experiments demonstrate that it is possible to radiate selectively the eyes of rabbits. The radiation cataracts that are produced develop at different rates and to different degrees, depending on the extent and localization of the radiation.

Mature cataracts were produced only in eyes that received total irradiation. The

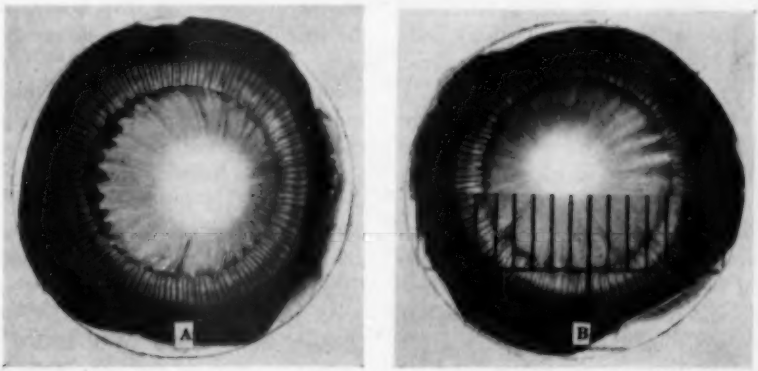


Fig. 4 (Puntenney and Shoch). Gross appearance of hemorrhages in ciliary body 48 hours after irradiation. (A) Whole eye. (B) Through peripheral aperture.

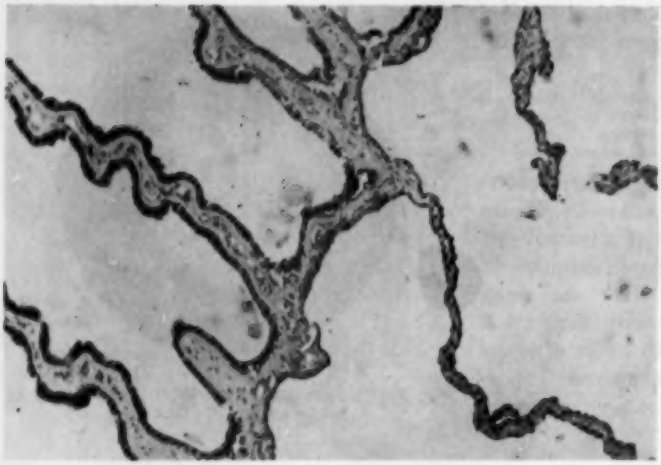

Fig. 5 (Puntenney and Shoch). Microscopic appearance of normal ciliary body sectioned parallel to plane of iris.

cataracts that developed in the other animals were characterized by peripheral changes consisting of vacuoles or granules or both, and these changes were confined to the subcapsular cortex.

Their incidence was closely related to radiation that was directed to the equatorial portion of the lens. This is demonstrated by the small percentage of eyes (15 percent) that developed lens opacities after being irradiated through a central aperture in a lead "contact lens."

When the periphery of the lens was radiated, 100 percent of the eyes developed peripheral opacities. The same percent (100 percent) occurred in the group irradiated with one half of the lens and ciliary body unshielded. Here the opacities were confined to the area radiated.

One of the most interesting findings is the very small percentage of eyes with lens changes in the group in which radiation was confined to the ciliary body. Only in Group III-C where the whole eye except the lens was exposed to radiation did opacities develop and here we may assume that there was some spread around the disc that did not occur in Group III-B when only a 1.0-mm. aperture between the central and peripheral lead shields was exposed.

The significance of this finding is not at first apparent, because the figures suggest that the ciliary body does not contribute to the formation of cataracts. The clue is found in the formation of mature cataracts in the eyes in which all the lens and ciliary body were irradiated.

In all the other eyes, either the lens or ciliary body was shielded, and the cataracts that developed were of the incipient type, with granular opacities and vacuoles confined to small areas in the subcapsular cortex.

This suggests that the initial injury takes place in the epithelium of the equatorial portion of the lens. The exact nature of the ionizing injury is unknown, but the extent of the lens damage must depend on the ability of the epithelium to survive. The events that occur between the absorption of the ionizing radiation and the biologic effect are probably chemical in nature, and here the presence or absence of a normal aqueous may have a profound effect in modifying the damage done to the epithelium of the lens and in maintaining the viability of both the old and the newly formed fibers.

As long as the ciliary body is not too severely injured, it may be said to exert a protective effect on the lens. In the group of eyes with the half-shield protection, one half

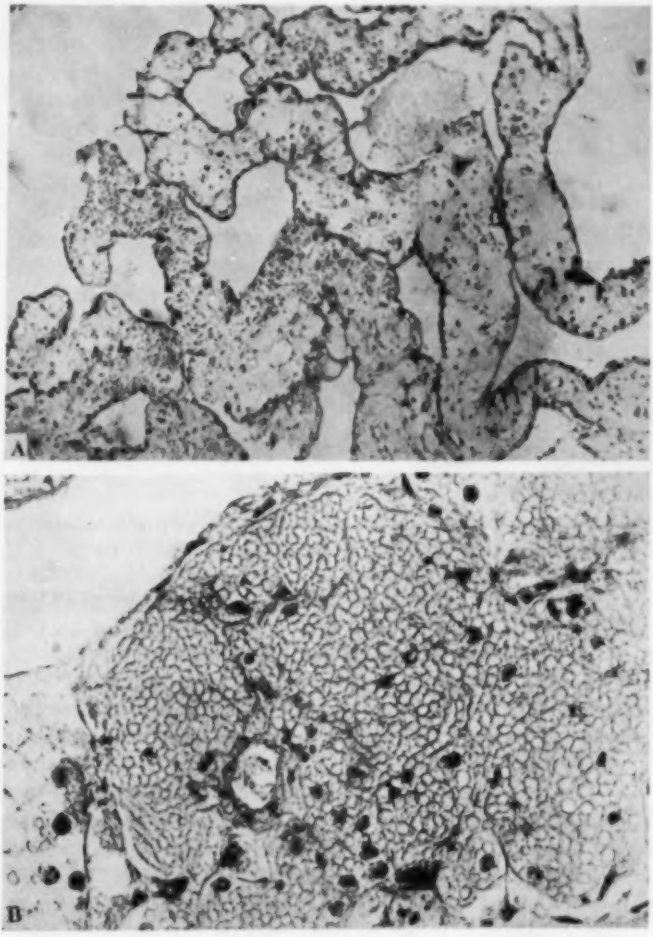


Fig. 6 (Puntenney and Shoch). Microscopic appearance of ciliary body 48 hours after irradiation. (A) Low power, showing hemorrhages, exudate, and edema. (B) High power, showing edema and leukocytes.

of the ciliary body was injured, and yet mature cataracts did not develop in five months. Apparently enough viable ciliary body was left to promote recovery of an injured lens.

Zirkle[12] and others believe that radiation produces an inhibition of cell division associated with quantitative changes in normal cellular processes. If radiation disrupts normal cell division, then the first lenticular cells to die must be those in mitosis or in a premitotic stage.

Poppe[13] has shown that mitosis is rarely seen in the normal lens, and this offers an explanation for the relatively few numbers of cells that are fatally damaged during the period immediately following radiation.

The various degrees of damage that occur in other cells may ultimately lead to further cell destruction, depending on the presence and level of the necessary aqueous constituents. Whatever the mechanism, there is an increased susceptibility to mature cataract formation when both the ciliary body and the lens are injured.

It is interesting to note that the incidence

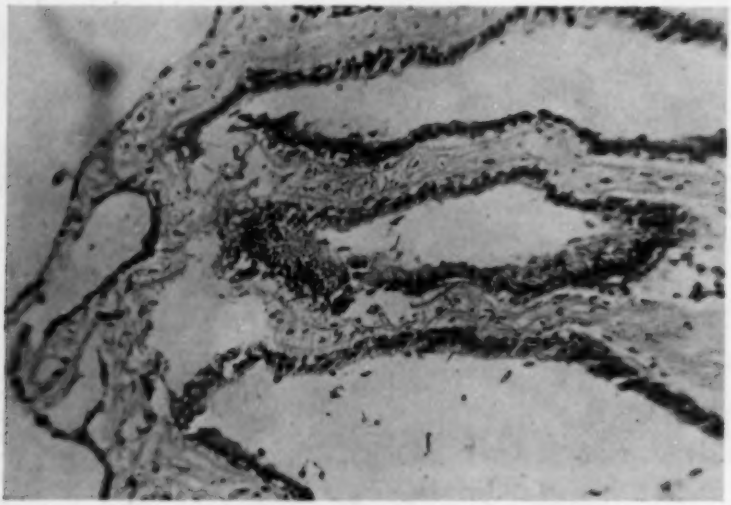

Fig. 7 (Puntenney and Shoch). Microscopic appearance of ciliary process five months after irradiation of whole eye. Slight atrophy and vacuolization of epithelium are present.

of cataract formation as presented in this report parallels to a marked degree the recently reported findings of Alter and Leinfelder[10] on the effect of shielding the lens and ciliary body. Essentially the same techniques and procedures were employed, except for the difference in the dosage of X rays given.

Alter and Leinfelder have concluded from their experiments that radiation cataract is secondary to an injury to the actively growing portion of the lens, the lens epithelium and the equatorial fibers being especially sensitive to roentgen rays, and that exposure of this region yields the same results as exposure of the whole lens.

An early study by Goldman and Liechti,[14] which makes this same point, was brought to our attention by Dr. Cogan at the time of the original presentation of this paper. By using a very fine beam of X rays and dusting a fluorescent powder on the cornea, they were able to confine their radiation to either the periphery or the center of the lens. The periphery was irradiated via an intact iris and via an iridectomy. They showed that only with peripheral radiation did lens damage occur and this was independent of the presence or absence of the iris.

The work presented here is in agreement with the findings of Alter and Leinfelder. However, a dose of 2,000 r is too small to produce maximum lens damage consistently; 4,000 r will consistently produce mature cataracts in four to five months if the whole eye is irradiated. When the periphery of the lens is irradiated, even with this large dose only incipient opacities develop. It seems reasonable to conclude that the initial damage is due to direct hits on the lens epithelium, and progression to mature cataracts is dependent on damage to the ciliary body.

The destructive changes that occur in the ciliary body can be demonstrated anatomically and by permeability studies. The anatomic changes are visible grossly in the acute stage; they consist of hemorrhages and edema. Microscopically the changes can be traced through the acute, subacute, and late stages.

In the acute stage, red blood cells can be seen scattered throughout the ciliary processes; this is associated with edema, distortion of the epithelium, actual destruction of

the cellular epithelium, and leukocytic infiltration. This also occurs when the lens is shielded and the ciliary body irradiated through a 1.0-mm. ring. The injury is more extensive when the whole eye is irradiated.

In the subacute stage, the reaction diminishes considerably.

In the late stage, many of the ciliary processes look fairly normal while others show atrophy, vacuolization of the epithelium, and areas of unabsorbed exudate.

The alteration in permeability to fluorescein, sodium, and potassium has been demonstrated by von Sallmann. Similar studies by us, using radioactive iodoalbumin, have corroborated his findings, and will be published elsewhere.

## Conclusion

1. Irradiation of the eye of the rabbit (4,000 r) produces granules and vacuoles in the crystalline lens in six to seven weeks. Ninety percent of these lenses proceed to complete opacification in three to five months.

2. Irradiation of the lens exc'usive of the epithelium does not result in cataract formation.

3. Irradiation of the epithelium of the lens alone produces cataracts uniformly in 40 to 50 days. However, these opacities remain stationary and do not go on to maturity.

4. Irradiation of the ciliary body alone does not result in cataract formation.

5. Irradiation of the whole eye or ciliary body alone results in transient gross and microscopic hemorrhages, edema, necrosis, and epithelial destruction in the ciliary body.

## Summary

An investigation has been made on the relationship of the ciliary body to radiation cataract. The eyes of rabbits were selectively irradiated through lead shields of different apertures. It was found that 4,000 r produced incipient opacities when only the lens epithelium was irradiated. When the lens epithelium and ciliary body were irradiated the incipient opacities progressed to maturity. Ciliary body irradiation alone did not produce cataracts. Gross and microscopic preparations were made which demonstrated destructive effects of X rays on this tissue.

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# THE PTERYGIUM: ITS ETIOLOGY AND TREATMENT

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The etiology or, in more precise words, the etiopathology of the pterygium has long been the subject of interest, and debate, between ophthalmic surgeons. Even now there is no general agreement about how the pterygium is caused and what factors underlie its formation, though the inflammatory basis, which I started to advocate in 1940, is gaining more and more approval.

The theories of the etiology of the pterygium are many and varied. The disease was known since the time of Hippocrates and, for a long time, was considered a type of pannus.

The first ophthalmologist to treat pterygium extensively was Susruta, thousands of years before Christ. He believed, and his belief is shared and elaborated by more recent writers (Bidyadhar), that formation of pterygium depends on the nutritional state of the individual and that it may be prevented by improving corneal nutrition by improving the general health of the patient. He advocated revitaminization of the eye by means of a diet of milk and fruit juice and advised treatment of nasal and dental infection.

Morax attempted to establish that the pterygium is one of the manifestations of lymphogranuloma, but in view of the negative Frei reactions obtained, he rejected the idea. Coutts, Lugue, and Hewitt, on the other hand, still insist on the lymphogranuloma theory. They base their belief on the observation that some granular bodies are found in the cytoplasm, as well as extracellularly, of the epithelial cells in cases of pterygium, similar to those seen in cases of lymphogranuloma. Also, one of their patients who had contracted lymphogranuloma by the genital route four years previously, developed a bilateral pterygium.

Doherty believes the pterygium is an expression of the need of the cornea for protection from external irritation, or, in his own words, "a call to Nature to supply a nictitating membrane corresponding to the palpebra tertia of animals."

Beard and Dimitry report that the tissue of the pterygium, when boiled with chloroform, gives a faint cholesterol reaction, which is not precipitated with digitonin. The application of choline chloride to the pterygium dilates the vessels which become greatly engorged with blood; at the same time, the fatty substances present in the tissues disappear. Formation and transportation of phospholipids may occur here, as is the case when choline carries on its lipotropic function by removing fatty material from the liver. On the basis of such findings, the two authors, who are not in favor of the irritation theory, wonder whether the formation of the pterygium is due to a choline deficiency in adults.

Michel Gerundo, in a recent publication, isolated Morax-Axenfeld bacillus from three out of 25 cases of pterygia. Since the three cases were in the early proliferative state, he could not consider the presence of the bacillus to be a simple coincidence and states "it is possible that the bacillus stimulated a chronic inflammation in an eye which was already irritated by dust and glare."

These are some of the etiologic theories of the formation of the pterygium. Many others

could be found in the literature. But the theories which I am going to discuss are only three: (1) the neoplastic theory, (2) the degenerative theory, and (3) the inflammatory theory.

### The neoplastic theory

The neoplastic theory of the etiology of the pterygium is advocated by Schreiter, Williamson-Noble, John McReynolds, and others. Many facts stand against this theory:

1. There is nothing in the histopathology of pterygia which indicates or alludes to a neoplastic origin or tendency.

2. It is very strange for a tumor to have the tendency to extend only in one direction and that on the avascular cornea.

3. If the condition were neoplastic in origin, the pterygium would continue to extend when tucked under the conjunctiva in the well-known McReynold's operation. Nothing of the sort happens.

In my opinion, since the neoplastic theory has many facts against it, it should be dropped from consideration as a probable cause of pterygium.

### The degenerative theory

The degenerative theory of the etiology of the pterygium defended by Schoninger, Fuchs, Parsons, Duke-Elder, and many others, points to the pinguecula as the precursor and considers the pterygium a further step in the same primary degenerative pathologic process. It is based on two observations:

1. The constant finding in all cases of pingueculas and of pterygia examined histopathologically of early signs of hyaline and elastic-tissue degeneration in the deeper parts of the tissues.

2. Pterygia frequently occur in cases in which a pinguecula is present.

Both observations are quite true. But is the pinguecula the cause of the pterygium and is the degeneration, whether primary or otherwise, the cause of both of them?

Degeneration is a term that includes all visible alterations in living cells (apart from simple atrophy) which are produced by disordered nutrition and present damage short of actual death. This disordered nutrition may be due to many and varied factors but, whatever the cause, the histopathology of the resulting degeneration is the same and the type of degeneration bears no relation to the causative agent.

Thus, a single cell with hyaline degeneration due to the effect of a toxin cannot, from its histopathologic picture, be differentiated from another cell that is affected with a senile hyaline degeneration or primary hyaline degeneration. The cause of the degeneration is not determined from the look of the cell affected but from the accompanying pathologic signs and clinical symptoms. When these facts are applied to the histopathologic and clinical pictures of the pterygium, it seems clear that the degeneration that occurs in the deeper parts of the tissues of the pterygium cannot be primary in nature; they must be postinflammatory for the following reasons:

1. The constant presence of round-cell infiltration in the superficial strata of the cornea in all cases of young and progressive pterygia. Round-cell infiltration is a pathologic accompaniment of chronic inflammations and not of primary degeneration.

2. The marked increase of the goblet cells of the conjunctiva in cases of pterygia. Goblet cells increase with chronic inflammations and not with primary degeneration.

3. Deposition of dense fibrous and connective tissue in the submucosa of the conjunctiva and between Bowman's membrane and the epithelium of the cornea which cannot be the result of a degenerative condition for there is no known degenerative condition that leads to formation of subepithelial fibrous tissue. Only chronic inflammatory conditions lead to damage and disappearance of the specialized tissues and to overgrowth of the more resistant connective-tissue cells. Replacement fibrous tissue is the response to

chronic inflammation and not to degeneration.

4. There is no known degenerative condition starting in a vascular and well-nourished area, such as the limbus, that tends to be self-limiting as it progresses toward the nonvascular center of the cornea. I have seen hundreds of pterygia of different types and in different stages of development and only a minute number ever reached the center of the cornea; none was ever seen to extend beyond this point.

5. The vascularity of the pterygium, the recurring periods of more acute inflammatory engorgement, the age of the patients, the pterygium's fixed position in the most exposed part of the bulbar conjunctiva—all favor an inflammatory etiology.

### The inflammatory theory

The inflammatory theory, which I began advocating in 1940, is based on clinical observations and pathologic facts.

At that time I was working in a locality where there were many cases of pterygia and I noticed in many cases that came to the hospital a special type of congestion of the conjunctival vessels in the area exposed in the palpebral fissure, mostly nasally but sometimes also temporally, which caused no discomfort to the patient, who had come to the hospital for something else.

On close examination, one finds an area of congestion, more intense near the caruncle, restricted solely to the part of conjunctiva exposed in the palpebral fissure, with vessels running radially toward the cornea but stopping short at varying distances from the limbus. The conjunctiva of the affected area is slightly thickened, more raised, and less transparent than the surrounding areas. In some cases the vessels reach the limbus.

Such a type of congestion, thickening, and loss of transparency in a restricted area of the bulbar conjunctiva exposed in the palpebral fissure is not known to have any relation to trachoma, the most common chronic conjunctival affection in Egypt. Also the severity of the congestion had no relation to the stage of trachoma. Some severe cases accompanied smooth trachoma IV and some mild cases were found with active trachoma.

In view of the fact that such chronic inflammatory cases were numerous and since they had never been described as separate clinical or pathologic entities, I excised some of the affected tissue for histopathologic examination. In all cases examined, the histologic picture was almost identical.

The epithelium was thinned; the nuclei of the submucosa had, for the most part, disappeared; there was hyaline and elastic-tissuelike degeneration which affected the newly formed connective tissue bundles in the submucosa. In many of the cases, goblet cells were increased in number. This picture, as can be seen, is identical in its basic features to the histopathologic picture of the fold of conjunctiva that forms a pterygium. Yet, clinically, in these cases there was no pterygium and the part of the conjunctiva affected did not show the slightest tendency toward pterygium formation.

Since I could not consider this close resemblance between the histopathologic pictures of the two conditions a mere coincidence, I started to follow these cases (which I provisionally called chronic irritative exposure conjunctivitis). When followed for a sufficiently long time it was found that:

1. Some of the cases remained stationary.
2. Others subsided without leaving a trace.
3. On subsiding, a pinguecula was left near the limbus in other cases.
4. In this group, a pterygium developed during a period varying between three months and one year. The development of the pterygium was preceded by the appearance of a haze formed of minute dots in the cornea at the limbus. This was followed by limbal vascularization extending from the congested vessels in the area of conjunctiva affected. As the haze of infiltration progressed toward the center of the cornea, the

vascularization followed and, at a certain stage, the conjunctiva itself began to encroach. The whole process was very slow, taking months to develop.

It became evident to me that the pathologic resemblance between the pterygium and the condition I called chronic irritative exposure conjunctivitis was not a mere coincidence and that either the second condition is the precursor of the first or that both are caused by one and the same etiologic factor. Because many cases of chronic irritative exposure conjunctivitis remained stationary or subsided without forming a pterygium, and because the pterygium was preceded by signs of limbal inflammation, the second deduction seemed more logical.

### Histopathologic study

The second step in the investigation was to learn the mechanism underlying the process of encroachment of conjunctiva over the cornea. So many inflammatory conditions, chronic and acute, of the cornea occur at the limbus (none of which show an encroachment of conjunctiva over the cornea) that, to clear this point, I took specimens from many cases of pterygia in different stages of development for histopathologic examination, hoping to find a difference between the pathologic pictures of early cases and late cases of pterygia—a difference that may account for, or explain, the mechanism of the encroachment. I could find such a difference and show how the conjunctiva creeps over the cornea in the formation of the disease entity we call pterygium.

In one group of specimens, four pterygia were present—two were well developed and advanced; the other two were very early (budding) pterygia.

The pathologic report on the two well-developed cases reads:*

Fig. 1 (Kamel). Section from a well-developed pterygium.

"The nuclei of the submucosa for the greater part have disappeared and the connective-tissue bundles have become confluent with each other and look homogeneously pink (hyaline degeneration). In the deeper part of this hyaline tissue, elastic-tissuelike degeneration is seen in the form of dark-staining convoluted fibers. The epithelium over the affected area is thinned." (See Figures 1 and 2.)

The report on the two cases of very early pterygia, was:

"The appearance of the specimens is exactly the same as those seen in your other two specimens but the elastic-tissuelike degenerated fibers are seen in cross section."

This difference in the appearance of the

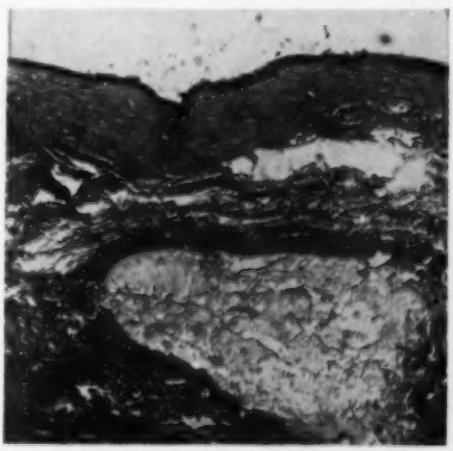

Fig. 2 (Kamel). Section from a well-developed pterygium.

* All my pathologic specimens were kindly examined and reported upon in the Giza Memorial Ophthalmic Laboratory.

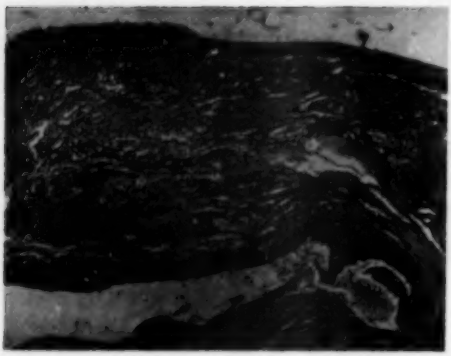

Fig. 3 (Kamel). Section from a "budding" pterygium.

degenerated fibers is the only difference from the histopathologic point of view, between the early and late pterygia. However, it is the difference that explains how the pterygium is formed. (See Figures 3 and 4.)

For, if straight horizontally disposed fibers are found and they are cut at right angles to their axes, the section will show the fibers in cross section. If, however, the same horizontally disposed fibers shrink and contract and then are cut at right angles to their axes, the sections will show the fibers not in cross section, but confluent and convoluted. For these reasons the pterygium is, in my opinion, an inflammatory condition.

Every inflammatory process either heals by complete resolution or, if the process lasts long enough, there is damage and disappearance of the specialized elements and their replacement by the deposition of the more resistant connective-tissue cells. A replacement fibrous tissue is the response of tissues to chronic inflammations. This is a pathologic fact and the conjunctiva and cornea are no exception to the rule.

As a result of the chronic inflammatory process that leads to the formation of the pterygium, fibrous tissue is laid down in the form of horizontal bands. It is laid in the form of horizontal bands, most probably, because it follows the radial disposition of the blood vessels of the conjunctiva, from the intima of which the fibroblasts develop. These fibrous-tissue bands, as all fibrous-tissue bands, will in time shrink and contract.

If the inflammatory process is restricted to the conjunctiva and the condition is only a chronic irritative exposure conjunctivitis, the shrinkage of the subepithelial fibrous-tissue bands will cause no harm. If, however, the conjunctival inflammatory process extends to the cornea and the chronic conjunctivitis becomes a keratoconjunctivitis, the shrinkage of the subepithelial, horizontally disposed fibrous-tissue bands, present both in the conjunctiva and cornea, will lead to the formation of the pterygium.

These bands adhere simultaneously to both the conjunctiva and cornea and, when they shrink, a certain amount of mechanical pull is exerted, which either pulls the cornea toward the conjunctiva or the conjunctiva toward the cornea and, since the cornea is fixed and the conjunctiva is more or less mobile, this pulling effect will cause the encroachment of the triangular fold of conjunctiva over the cornea—the condition we call a pterygium. The more active and progressive the case, the greater the amount of fibrous tissue which will be laid down and, because of contraction, the farther the pterygium will progress toward the center of the cornea.

The pterygium, in my opinion, is, therefore, the symptom of an inflammatory process that starts in the conjunctiva as a chronic irritative exposure conjunctivitis.

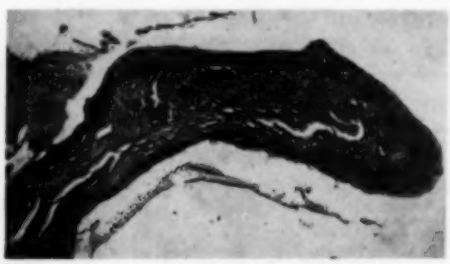

Fig. 4 (Kamel). Section from a "budding" pterygium.

The condition thus started may follow one of two courses:

1. It may be restricted to the conjunctiva. If this occurs, the result is a condition which I call chronic irritative exposure conjunctivitis and which resembles, clinically and pathologically, the fold of conjunctiva that forms the pterygium.

2. If, on the other hand, the chronic inflammation extends to the cornea and the chronic conjunctivitis becomes a chronic keratoconjunctivitis and results in the shrinkage of the subepithelial fibrous-tissue bands, a pterygium is formed.

A pterygium is, according to my theory, neither a disease of the conjunctiva nor of the cornea; in fact, it is not a disease at all. It is only the result of an underlying inflammatory process of which the pterygium does not become a symptom, unless both the conjunctiva and cornea are affected simultaneously. It is my opinion that the definition of the pterygium should be:

"It is a mechanical encroachment over the cornea of that part of the bulbar conjunctiva exposed in the palpebral fissure as a result of the shrinkage and contraction of radially disposed fibrous-tissue bands laid down in the submucosa of the conjunctiva and cornea in response to a chronic irritative exposure keratoconjunctivitis."

## Treatment

When I wrote my first paper, in 1940, advocating the inflammatory theory of the etiology of the pterygium, I presented my operation, based on the theory, for the treatment of the condition and which had been successful in more than 600 cases. Surgery of the cornea at that time was looked upon with some misapprehension; nevertheless, the operative procedure, which I then advocated, was accepted, in Egypt and abroad, with gratifying results. This original operative procedure has been slightly modified, without affecting its basic principle, in the light of the many advances in medicine and surgery during the last 13 years.

The treatment of pterygium which I now use depends on whether the condition is recurrent or primary.

### Primary pterygium

When there has been no previous operation, the surgery is as follows:

The conjunctiva is anesthetized with drops of percaine with adrenalin. The cornea is then marked with a sharp knife about one mm. away from and around the advancing head of the pterygium, the mark being stained with fluorescein. The central border of the marked cornea is then slightly undermined with the knife and the edge held with a fine forceps.

A partial lamellar keratectomy of that part of the cornea which is marked, with the head of the pterygium included, is then done, either using an old Graefe knife for the dissection of the lamellae, or, better still, using Castroviejo's utility scissors. The dissection is carried to the limbus and the conjunctiva is undermined till the caruncle, or the outer canthus, is reached. Great care is taken not to leave adherent parts of the conjunctiva to the cornea, up and down, at the junction of the neck of the pterygium with its body.

In cases in which there has been no previous operative interference, the conjunctiva is thin, with no adhesions to the sclera and no appreciable amount of subepithelial fibrous tissue that can be dissected separately. All fibrous tissue will adhere to the under surface of the undermined conjunctiva.

The corneal lamellae, with the head of the pterygium, is then snipped off and the conjunctiva is held with forceps away from the sclera while its under surface is cauterized with carbolic acid. This finishes the operation.

An antibiotic ointment is applied and the eye is bandaged. No stitches are needed because there is no appreciable amount of bare area left behind.

When the head of the pterygium, with the corneal lamellae, is excised, the conjunctiva is seen to retract for a few mm. from the limbus. This area is quickly epithelized within a few days.

The cornea is dissected in the manner of a partial lamellar keratectomy because this leaves no scar behind. When the head alone is dissected from the cornea, as I did at first, islets of adhesion may be left behind. These are undesirable from the cosmetic point of view.

The under surface of the conjunctiva is cauterized with carbolic acid to enhance the healing process. On the second day, the wound is dressed with antiseptic drops, antibiotic ointment, and cortisone ointment. This continues for five days, after which the bandage is discarded. Cortisone is applied to prevent any reaction in the cornea and to prevent or delay the formation of fibrous tissue until complete epithelization occurs.

### Recurrent pterygium

In cases of recurrent pterygia, dense fibrous tissue is usually present under the epithelium and the technique of the operation must accordingly, be modified:

The conjunctiva is anesthetized and an incision is made concentric with the limbus and slightly more than the width of the pterygium head. Then the conjunctiva alone is held with forceps and dissected and undermined till the caruncle is reached, leaving the head of the pterygium still attached to the cornea and leaving the subepithelial fibrous tissue in place.

A superficial partial lamellar keratectomy is then done, as in the previous operation, including the head of the pterygium. When the limbus is reached, the fibrous tissue continuous with the head of the pterygium is dissected from its attachment to the sclera. Now, the subepithelial fibrous tissue and the head of the pterygium are together in one sheet, all of which is excised.

The under surface of the conjunctiva is then cauterized with carbolic acid and the operation is finished as in the operative procedure described for primary pterygia. The only difference between the two techniques is that, in one, an appreciable amount of subepithelial fibrous tissue is excised and removed; in the other, this is not done.

This is my present treatment for pterygia. It does not differ in its principle from the operation previously advocated but the new corneal technique and the application of cortisone and antibiotics make the results certain and the cosmetic effects better.

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# A TECHNIQUE OF CORNEAL GRAFTING DEVELOPED IN NEW ZEALAND

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The following method of corneal grafting is one that I have developed and been using in part or in whole since 1947. I have found the technique simple and the results uniformly good.

I have always felt that better results and higher percentages of clear grafts in keratoplasty must come largely from improved technique. It has been my endeavor to obtain a cleanly cut disc of cornea to fit exactly a cleanly cut hole in the recipient cornea. This should assist rapid sealing of the wound, thus helping to avoid complications such as anterior synechias, and should lead to the minimum of opacity from scarring when the wound heals.

The making of a cleanly cut disc from the donor cornea has been solved by Amsler and Verrey and I always cut my graft in this way. The excised cornea is placed epithelial side down on a block of hard paraffin and the disc punched out by pressing a trephine through it without rotation.

Cutting a clean disc from the recipient cornea however is a different problem. I found it difficult to cut the disc completely with either a hand or mechanical trephine. In fact I only succeeded in doing it once during an actual operation and that was with an electric trephine developed and made here by Mr. Frost to my requirements. Also there is always the fear, and no doubt some risk, of damaging the lens or iris with the trephine. In the majority of cases, therefore, when the trephine has entered the anterior chamber in one place, the section has to be completed with scissors which usually leaves some irregularity or shelf of corneal tissue posteriorly. This cutting a clean disc from the recipient cornea has been achieved by a special punch which I shall describe later.

The graft is fixed in position by a corneal splint which also helps to seal the whole wound against loss of aqueous.

## Corneal splint and trephine guide (fig. 1)

My corneal splint and trephine guide was fully described in the *British Journal of Ophthalmology*, September, 1949.

The guide is made of an inert metal such as tantalum. It is molded to fit the convex surface of the cornea. It has a central aperture just large enough to admit the particular-sized trephine chosen for the operation. From the circumference, four arms project a distance to fall just short of the limbus. At the end of each arm, notches are cut to be held by corneal sutures.

The splint is a replica of the trephine guide except that the hole is smaller so that the splint will overlap the circumference of the graft as well as part of the surrounding recipient cornea.

At operation the guide, which assures accurate centering, is first placed on the cornea in the appropriate position while four sutures are passed to take a good bite of the cornea at the end of each arm. These are tied once over each arm without completing the knots. This is sufficient to hold the guide in position

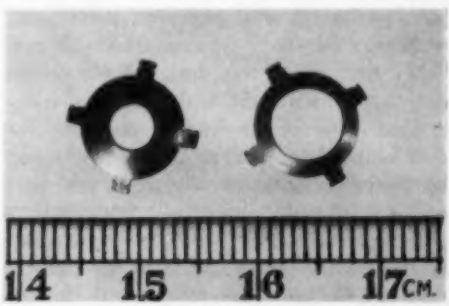


Fig. 1 (Pittar). Corneal splint and trephine guide (enlarged).

while the trephine is rotated several times through the central hole of the guide, thus marking the corneal disc to be removed. The suture loops are then loosened without undoing completely and the guide removed.

When the corneal disc has been removed and the graft introduced, the splint is immediately placed in position and the suture loops tightened over its arms and the reef knots completed. This gives even support to the graft all round and, as the reforming aqueous presses the graft and surrounding cornea forward against the splint, the latter tends to seal off the wound against loss of aqueous.

Stallard suggested a modification of the splint making it of clear acrylic with no central aperture so that it covers the whole of the graft. I have found this equally satisfactory, its only disadvantage being that it cannot be boiled for sterlization.

More recently Philps and Fincham have described a plastic corneal splint which covers practically the whole of the cornea being held in place by sutures through peripheral holes instead of having arms. This does not require the use of a trephine guide. I have not tried this particular splint but of course the principle of fixing the graft is exactly the same as with my original splint.

## The corneal punch

During the development of the punch, it was realized that the ideal was that the whole circumference of the corneal disc should be punched out in one piece for perfect accuracy.

My first punch was designed for use in conjunction with the orthodox trephining method. The object was to complete the removal of the corneal disc, after trephining, by a punch instead of scissors to get more accurate sharply cut edges without any shelving posteriorly. Naturally the circumference of the punch had to be the same as that of trephine. Therefore, unless the trephine perforated the cornea for at least half of its circumference the punch could not be introduced into the anterior chamber. In such a case, scissors still had to be used at least until the punch could be introduced.

A different approach to this problem then occurred to me. A trephine was merely used to mark the cornea, the superficial cut being stained with fluorescein. Then, with a narrow Graefe knife, I cut right across the diameter of this ring after the manner of a Saemisch section. It was then possible to introduce the punch and remove first one half of the disc and then the other. I did several cases by this method quite successfully but there were still chances of imperfections in the bed thus formed. It was not easy to be certain, on closing the jaws of the punch, that one was exactly on the line marked out. This difficulty was increased by the fact that the Saemisch section may be slightly longer than the diameter of the circle thus allowing the possiblity of slight horizontal error in placing the punch as well as vertical error.

These experiences led to the modification of the technique and instrument which I first described at the Congress of the Ophthalmological Society of New Zealand, September, 1952.

### Description of corneal punch (fig. 2)

The punch takes the form of a stainless steel tube of the appropriate diameter. In this tube slides a perfectly fitting piston. A portion of the lower part of the piston is cut away right around its circumference to leave a mushroomlike end attached to the main body of the piston by an axial "stalk." There is a vertical slit in the tube through which an arm can pass into a hole bored in the upper part of the piston to move it up and down. So that the tube will be of sufficient strength to prevent distortion by the location screw, a short thick-walled bush is press-fitted into the upper end of the thin-walled die tube.

A pair of forceps has been adapted to work the punch. The ratchet lock has been removed and the forceps' nose shortened to give as great a leverage as possible without

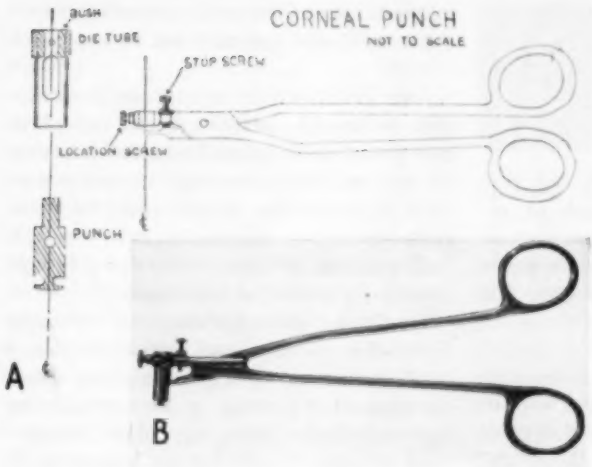


Fig. 2 (Pittar). (A) Drawing of design of punch. (B) Photograph of punch.

spring and without requiring too great a spread of the hand. On one arm of the forceps has been brazed a light-weight stainless steel collar which holds the upper end of the tube, the location of which is secured by a set screw. The other arm has a ball-shaped end which fits into the piston through the slit in the tube. Thus opening and shutting the forceps moves the plunger up and down.

The punches can, of course, be made in any diameter. I have them in 5.0, 5.5, 6.0, 6.5, and 7.0 mm. They all fit the same handle and can be changed merely by manipulating the location thumb screw.

## Construction

### Steel

The steel selected was Commonwealth cutlery quality G-2 stainless steel made in Australia by Brokenhill Proprietary, Ltd. During the steel's annealed form, the punch parts were turned, bored, and polished on a nine-inch precision lathe leaving small amounts for grinding in die hole, outside of bush, and outside of punch. Because of the work hardening properties of this steel soya bean oil is an essential lubricant in drilling.

The slot in the punch was cut by using a single fluke D-bit end-mill.

### Heat treatment

The parts were then carefully wired together and placed in a neutral salt bath furnace where they were slowly raised in temperature to 800° C. then quickly to 960° C. and quenched in oil. Tempering was at 200° C., giving a Rockwell hardness of approximately 60.

### Grinding

Grinding is very difficult on stainless steel of such small diameters as no spark and no noise is given off to indicate when grinding is taking place. The parts were held in an infinitely variable Jacobs collet chuck which provided the necessary accuracy. The die tube was ground with an included angle of 120 degrees, leaving a 0.002-inch shoulder for cutting. Clearance between punch and die tube is 0.0002 inch.

### Lapping

The final operation was lapping and consisted of a copper lap turned to the correct size and charged with lapping compound, great care being taken while lapping not to allow any bellmouthing of the die tube. Likewise the utmost care was taken to protect the correct size of the head of the mushroom punch.

### Testing

After assembly each punch was tested for cutting ability on 0.001-inch thick cigarette paper, a severe test for any punch.

### Conclusion

With all instruments of this type the greatest care and absolute precision are required at all stages of manufacture. To protect this accuracy the greatest care must be exercised at all times when handling this instrument.

### The operation

After the cornea has been marked with the trephine and guide, a knife section is made from within out by transfixing the cornea right across the diameter of the marked ring. It should enter and emerge just beyond the ring. For the left eye I prefer to stand below the patient's head on his right side and make the section with my right hand rather than use my left.

One edge of the punch is now slipped under the lower edge of the cut cornea and a piece punched out. Of course it will not reach to the edge of the marked ring in any direction because of the central post or stalk of the punch. Now a similar segment is punched from the upper lip of the sectioned cornea. It will then be found that by pushing one edge of the punch as far as it will go either up or down the whole mushroom end can be introduced into the anterior chamber, the stalk being in the central punched-out area. The punch is then centralized over the marked ring and the whole circumference of the corneal disc punched out in one piece (fig. 3).

The graft, which is prepared immediately before the operation is commenced, is now placed in position. The splint is placed on top and the previously formed suture loops tightened over the arms and the knots completed.

The splint is usually removed 14 days later under pentothal anesthesia.

In the last nine months since obtaining Grieshaber stainless steel corneal needles, I have done some of my cases using direct suturing of the graft to the surrounding cornea instead of using the splint. One gets very accurate closure but my impression is that if anything the splint cases do better and of course the technique with the splint is so much easier. I have therefore gone back to using the splint exclusively for fixation.

### Discussion

1. It will be seen that as the whole peripheral portion of the corneal disc is punched out by the accurately made punch in one motion there will be formed a truly circular bed with sharply cut edges.

2. If the punch is not perfectly centered over the marked ring when the final punching is made, it is of little importance. It merely means that the bed will be displaced a fraction of a millimeter in one direction. There is sufficient overlap of the splint to allow adequate coverage on the edges of the wound and graft. Again any such slight error

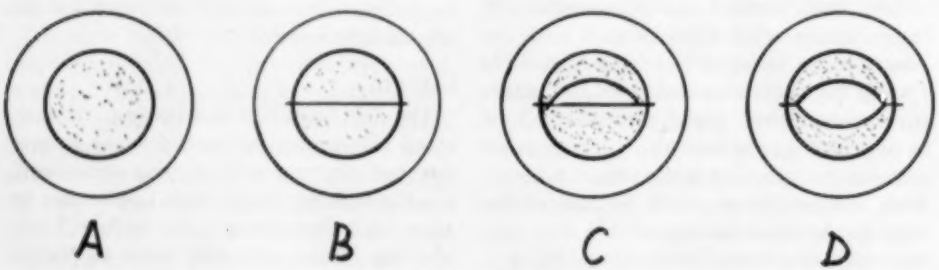


Fig. 3 (Pittar). Diagram of technique.

is unlikely to affect the visual result especially if one never does a graft smaller than five mm.

3. It does not matter if the Saemisch section projects beyond the edges of the bed as it is a perfect short lineal cut which has no tendency to gape and will be completely covered by the corneal splint. It is in fact necessary that the section should be very slightly greater than the diameter to allow for the thickness of the jaw of the punch which has to enter the anterior chamber. In making the section one must also remember the obliquity of the knife section and so allow for this as well by making the points of entry and exit of the knife outside the edge of the marked circle.

4. I believe that this is a simpler technique to carry out than the classic trephine method and that it removes that tension that a lot of us must feel while trephining due to the fear of damaging the lens. The making of the section is very simple, being very much easier than an ordinary cataract section.

5. It may be argued that if the corneal opacity is gross the section might be difficult because the knife cannot be seen in the anterior chamber. I can only say that this has not been my experience. In most cases the knife can be seen fairly well. However in cases where the point of the knife cannot be seen I have had no real difficulty in making the section. In such a short travel of the knife one seems able to gauge the counter puncture satisfactorily.

6. The main difficulty with the punches is to get them to cut completely. Even these latest ones are sometimes inclined to leave a bit of tissue undivided. I think this depends a good deal on the texture and condition of the cornea, for these diseased and scarred corneas vary considerably. Whether the punch can be still further refined by reducing the clearance of the plunger even more and thus get over this difficulty I do not know yet. However when it occurs it is not serious. It is merely a thread of tissue which can be easily severed with scissors. As will be seen by considering the clearance between the punch and die tube any undivided thread can only have a compressed thickness of 1/5,000 of an inch. I have found that the small preliminary segments of cornea are often not cut completely by the punch but that it nearly always cuts completely when punching a full circle.

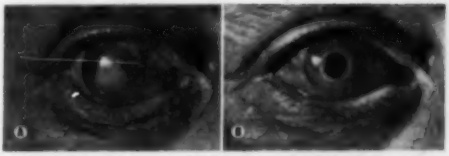

Fig. 4 (Pittar). A man, aged 28 years, had severe interstitial keratitis. (A) Before operation. (B) After operation.

### Case reports

Figure 4 is shown because it was an interesting case, illustrating a clear graft in a very opaque cornea. There was no difficulty in making the section although the knife could not be seen in the anterior chamber.

This was a man, aged 28 years, who had a very severe interstitial keratitis which left the cornea so opaque that neither pupil nor iris detail could be seen. He had perception of light only. Intraocular pressure was normal.

The worse eye was operated on. The section was made quite easily but the edge of the punch could not be pushed into the anterior chamber. I then found that there were anterior synechias due apparently to iris bombé adhering to the back of the cornea. These adhesions were separated with an iris repositor after which the disc could be punched out.

There was a complete pupillary membrane which I was able to strip off and remove entirely separating the posterior synechias. This revealed anterior cataract changes which can be seen in Figure 3 through the clear graft.

The graft (5.5 mm.) took well and re-

Fig. 5 (Pittar). A girl, aged 19 years, had corneal nebulae, mainly central. (A) Before operation. (B) After operation.

mained suprisingly clear. Vision however was only improved to counting fingers. Fundus details could not be seen except as a red reflex owing to the cataract changes. It is presumed, however, that there was retinal damage from choroiditis.

It seems to me that this would have been a far more difficult case to have done by the trephine method as the iris would have almost certainly been mutilated when removing the corneal disc. It is true that by the method I used it may have been punctured by the knife during section but, if so, it would have been just a clean radial incision.

Figure 5 is included because, in this case, I made a rather larger section than I intended. The fact that this does not matter is well illustrated, since this case did very well with an easy convalescence.

If you look carefully at the photograph at about the 3-o'clock position you can see a lineal scar of the section extending out from the circular scar. Note also the four scars near the limbus where the splint sutures were placed.

This was a 5.5 mm. graft in a Maori girl, aged 19 years. She had corneal nebulae mainly central, reducing vision in both eyes to counting fingers. There was a history of interstitial keratitis 13 years previously. She had a negative Wassermann reaction but was suffering from pulmonary tuberculosis.

Her operation was in November, 1951, and this photograph was taken two months after operation. Vision in this eye was 6/24 without glasses and with a correction for 5.0D. of astigmatism came up to 6/9.

In conclusion I wish to acknowledge the large part played in the development and production of this corneal punch by two men. They are Mr. Harry Frost, surgical instrument maker, and Mr. Eric Paton, precision engineer, both of Auckland.

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# ALBRECHT VON GRAEFE: THE MAN IN HIS TIME

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PART II*

On November 1, 1850, von Graefe, the young specialist, rented three small rooms up two flights of stairs on the Behrenstrasse and there he twiddled his thumbs for a few days while his instruments, bottles of silver nitrate, lead acetate and tincture of opium, his record books and prescription pads, all neatly laid out on a table and antique desk, gathered dust. "I need a larger sign," he decided at last uneasily. "The sign is too small."

"Not the sign," Wauldau offered. "The town, perhaps. Berlin itself may be too small for you and old Juengken at the same time, who thinks he can take care of all the eye work in the city with one hand tied behind his back, and Boehm, who imagines his book, *Squint and Tenotomy*, has said the last word on everything! According to gossip those two greatly resent the fact that you have had the temerity to open an eye clinic in a vicinity whose every ophthalmological need is being so brilliantly attended to already!"

The following day an advertisement began to appear in all the Berlin papers: "Dr. Albrecht von Graefe, in Behrenstrasse, will treat eye diseases free of charge for the poor." There was no mention of the rich, but they came too. The advertisement ran for six weeks.

Ten months later the 23-year-old physician had treated 1,900 patients and was beginning to look around for a place to move to, big enough to accommodate his practice and enlarged staff, and soon he found it at 46 Karlstrasse.

The substantial three-story dwelling, built on the corner, where the River Spree parts company with the Thiergarten, at no great distance from the "Charité," was more three houses than one, taking up an impressive footage along the fashionable thoroughfare. There the proud young lessee hoped to make room for a hundred hospital patients, besides having a clinical department, lecture and operating rooms.

It could not be done today, with one bath, three water closets, three sinks, and three cold-water faucets to the whole establishment including the kitchen, with candles or kerosene lamps for light—gas illumination would not be installed for another four years—and no central heating, but a century ago, the resolute young man saw no obstacles and "what the eye does not see and the mind does not know, does not exist."

During June and July the building was renovated, remodeled and fitted up according to the taste and needs of its new possessor, who never let a morning or evening go by without coming in to look around. It was easy to get lost in the many winding passages and stand confused before big double doors, when they happened to be shut, not knowing for sure which room was beyond, but one learned eventually. After a while the steep dark staircases did not seem so steep or so dark either.

The pleasant white-washed sick rooms, furnished with mahogany beds and chests, were small, but in each a double window, with a weather-blind of heavy linen twill moving between the two panes of glass, looked out upon the street in front or the small garden behind. Doors were edged with woolen cloth to keep drafts and noises out, chamber curtains, inner blinds, and bed screens were all of handsome dark blue French cashmere, folding candle screens of soft green silk. There was a glazed tile stove in every room

* The first part of this article appears on pages 525-543 of the October, 1954, issue of THE JOURNAL.

to keep it warm in winter. The floors had been left bare but it was supposed that certain wealthy and spoiled patients would bring or send out for carpets, along with other luxury gear they could not dispense with even for a few days.

On the ground floor were five rooms for private patients, five small cubicles making up the diphtheric ward, a fairly large clinical department, a porter's room, a three-room apartment for the inspector or overseer, two large rooms for patients to gather and wait in, a reception room that could also be used for operations or lectures, what was later to be an ophthalmoscopic room, and a room for examinations. Here instruments and records were to be kept, and conferences and consultations held.

Forty patients and guests could sit at table in the dining room, with a piano and a great crystal chandelier, next to which was the small "garden saloon," a kind of parlor, where coffee was to be served every afternoon at four. From here a flight of steps led down into *der kleine ziergarten*, a plot of green with a few shrubs about the edges.

The kitchen, to be presided over by a male cook, was in the cellar, with quarters nearby for the male servants of the establishment, while the majority of sick rooms were located on the first floor (what we in America call the second floor). There were a few single rooms for private patients on the top floor as well, but mostly it was given over to several wards, each furnished with a sofa and from three to six beds.

Some consternation was manifested at home and among old family friends at the way money was being spent like water on this new establishment. They knew where it was coming from, from his patrimony, that was where, and to dip into one's capital at so young an age seemed to invite disaster. What if his great scheme should fail? But they spoke of this among themselves only, not to him. If they had any notion that 15 beds on that top floor were to be occupied by charity patients, with meals and incidental expenses being paid out of the enthusiast's own pocket, they would have thrown up their hands in horror!

His mother said it was like *eine braut* furnishing her first house, to see the way Albrecht insisted on certain colors and fabrics, and the care with which he selected beds and linens, even pots, pans, and chinaware. It took two months to get the new place ready for occupancy and, when it was all ready to move into in early August, Graefe left for England, to attend the first International Medical Congress and see the Great Exhibition. Faithful Waldau, left in charge, was to supervise the clinic's removal from the Behrensstrasse to the new address on the Karlstrasse.

Graefe, upon arriving in London, did not stop to change the alpine clothes he had travelled in, but went directly to Guthrie's ophthalmologic hospital, where he was to meet Jäger the younger, from Vienna, who was operating there as guest surgeon that morning. Told where he could find his old acquaintance, whose demonstration was finished, he burst in upon him. A bystander took a hasty step backward out of his path and watched with some amusement how impetuously and with what delight the two men greeted each other.

For some time they were oblivious to all about them, but then Jäger caught a glimpse of the round smiling face behind his visitor. "Here," he said, "you two are made for each other!"

The man into whose arms he literally threw young Graefe was Frans Cornelis Donders, a 33-year-old professor from Utrecht, who blushed like a girl. He had chin whiskers that stretched from one earlobe to the other in the shape of a crescent moon, clear brown eyes, good features, and he was wearing the first new clothing he had had in more than a decade—a black broadcloth suit with coat to the knees, a checkered vest, white shirt and black silk cravat—bought for the occasion at the insistence of his wife who said that he was *not* going to

appear among all those fine people there in London looking like a vagabond. His salary was $400 a year! and he could easily afford it, Frau Donders said.

The tall handsome blue-eyed young man in alpine costume, with the soft short beard of clustering curls like a mediaeval baron's and the shoulder length dark hair he was to affect till the end of his life, this aristocrat who did not know what it felt like to be ill at ease, and the shy, unworldly professor in his stiff new suit, these two began with some hesitation to get acquainted.

Jäger knew well what he meant when he said they were "made for each other." Smilingly he watched them as they talked—as they sized each other up—and talked some more—and then some more—and soon, as he had known they would be, they were lost to Jäger and everybody else in the sweltering room.

Donders, born in Tilburg, Holland, was educated as an army surgeon, but did not pursue this career very long. Several years before, he had become interested in the mystery of human sight with its variations, *raison d'etre* and the hazards that might blot it out. In 1845, he became editor of the *Nederlandsch Lancet*, beginning at that time the intensive study of the muscae volitantes he was to carry on for two years.

In 1848, the year he joined the Utrecht Faculty as professor, he began to calculate the use of prismatic glasses in strabismus, examine the relation between convergence of visual axes and accommodation, and consider the regeneration of the cornea.

Far, far in the future was his glory, his great work on *The Anomalies of Refraction and Accommodation*, to be published not in Dutch but in English, the main source of knowledge about how to improve vision by the use of glasses, years distant the time when he would invent the ophthalmotonometer, and add to medical prolation the terms hypermetropia, ametropia, aphakia, and presbyopia.

Graefe, however, almost from the first moment they met that August morning, knew that here was someone, friendship with whom was to abide for 20 years, destined for a great fulfilment. Donders, for his part, in no time at all was convinced that the young German, for all his gayety and high spirits, laughter and jokes, was a man destined for immortal fame, consecrated, of divine gifts, almost certainly a genius. Neither was to be disappointed.

With pleasure, they discovered that they were staying under the same roof, as guests of William Bowman.

Again Graefe, as he had the year before, fared forth each morning from lovely Joldwynds into London's ophthalmological world and again returned each evening. But now he went arm-in-arm with Donders, ally and comrade and, if they two were happier those last days of summer than most other mortals, which none could doubt who saw them conversing heedless of all besides, it is not to be wondered at.

If they had not been put into a carriage by good-natured Bowman, who gave instructions that they were to be taken up Rotten Row and deposited at that entrance of the Crystal Palace, it is doubtful if they would have got around to visiting the Great Exhibition. And Donders, at least, would have got a sharp scolding when he returned home and had nothing to tell his curious wife and little boy and girl.

Generally a visitor fell silent with awe when first he stepped foot inside the great glass building, big enough to cover 18 acres of ground, with 16 semicircular ribs soaring to a height of 108 feet over his head. So improbable a place it was, with its red girders, slender yellow columns with sky-blue hollows, its transparent walls and vaulted roof, glittering with sunshine or, when "Queen's weather" did not prevail, shimmering with rain, its fountains, flowers, shrubs and great green trees (for many ancient elms had been enclosed within the building) that it struck most viewers dumb when gazed at for the first time.

So it might have done Graefe and his companion, but it so happened that, just as they were walking in, they touched upon the topic of tension in the eye and how to ascertain it accurately, and this was so interesting that somehow or other the moment of wonder passed without their taking cognizance of it.

They kept right on talking . . . but now they had to speak louder, listen more circumspectly and put their heads closer together, all of which they did unconsciously. They adjusted themselves by these refluent means to the novel contingency of having to promenade through an area crowded with thousands of people, all bent on seeing the greatest number of marvels from all over the world that had ever been gathered together in one place at one time, amidst the din of three tremendous organs ululating at once, but that they were adapting to what new circumstances they had no very clear idea.

Earls and duchesses, the Duke of Wellington himself, had been struck all of a heap by what they beheld here, and so might Albrecht von Graefe and Frans Cornelis Donders, only it befell that as they strolled around the building, one of them happened to mention the ophthalmoscope! and that started them off on a new track. How much longer were they going to have to wait for it? they wondered. Donders had tried to build such an instrument himself, he said—like this—he showed how it was put together—the reboation from the three organs was almost unbelievable—the two companions drew still closer together, Donders lifting his voice. But it hadn't worked very well. . . .

Thus discoursing, they passed Montgolier's great hydraulic ram, the distinguished undertaker, Mr. Shillibeer's, expanding hearse, a diving suit of caoutchouc, a boat designed so that it could be submerged and navigated under water, object of considerable merriment, as might be supposed. . . . They might have had their attention caught by the surgical counters had they not been sidetracked past that section by the milling crowd—Graefe was just then describing an embolus in the central artery of the eye, with anemic fundus and sudden loss of vision—but they were carried by all unaware and missed being astonished by the medical display. It was not until the yellowish statue of ivory Prometheus, padlocked to his rock with an ordinary padlock, began to seem like an old and suddenly very tiresome acquaintance to Graefe, that he realized they must have been round the pavilion three or possibly four times and had done all the "seeing" that ought to be expected of anybody, as he informed his companion. Donders expressed relief. His new boots had begun to feel like retribution for a long life of misdeeds. Standing, they ate a big Bath bun, drank a cupful of lemonade, and took their departure, feeling that they had done all that virtuous men could do for one day.

That evening, out on the terrace at Joldwynds, where hosts and some eight or nine invited guests had gathered before dinner to watch the sunset and sniff the roses, the two sightseers were asked what they had thought of the Fair.

Mrs. Bowman began it by inquiring if they had enjoyed themselves at the Exhibition? Oh, indeed, yes; yes, indeed. What did they think of the dais? she wondered. The dais? What dais? They looked at each other, Donders uneasily.

Why, they couldn't have missed that, she said, not even if they had tried! It had a canopy 40 feet high, a carpet made by a 150 women, and the chair of state had a red velvet mantle trimmed with gold thrown over it! Just behind it was a fluted silk tent and on either side equestrian statues of Victoria and Albert four times bigger than life!

"I *do* seem to remember something about statues," Donders said, wetting his lips.

"I must have missed the dais," Graefe confessed.

"They were there," Sir William announced to no one in particular. "I put them in the carriage myself."

"You can bring a horse to water—"

"We are *weary* enough to have been there," Donders offered apologetically. "Or at least I am."

"What was your favorite exhibit?"

"Why, aaah—"

"Let me see—"

Graefe and Donders exchanged desperate glances.

"Surely you couldn't have missed the refrigeratory? Box, you know—makes ice—keeps foodstuffs in a frozen state—they say Napoleon had one like it at St. Helena—"

"You must have smelled the olives from Portugal," old Dr. Wardrop put in pettishly.

There was a roar of laughter in which the two discomfited young foreigners joined.

"This should teach us two eye physicians something," Graefe said to Donders when he had recovered from his mirth enough to speak. "That it takes more to see with, than just a pair of eyes. . . ."

Waldau had discharged his trust nobly, the change had been made from the Behrensstrasse to the Karlstrasse without so much as a broken bottle; Graefe returned to find his new Polyclinic put in trim and ready to open for business.

On his desk, in that midst of three weeks' mail, he found something that made him sing out. "It's here! Helmholtz's paper on his ophthalmoscope!" When he had read it, he reached for his pen.

"Esteemed Professor . . . I hope you will pardon me, a complete stranger, for taking the liberty of addressing you and requesting a favor concerning a subject that interests me beyond anything. Last summer . . . in Vienna . . . I learned that you have made an instrument for examining the retina in the living eye. I later had . . . the pleasure of hearing . . . from Professor Brücke . . . some of the details of this accomplishment. The news of your triumph overawed us . . . I awaited . . . the publication with impatience . . . and now it is at hand. To the study of this article I am not only indebted for an exact understanding of the instrument but also for the explanation of other questions in optics which were formerly quite closed to me. I am, therefore, going to ask you if you will kindly request your optician to send one or two of your finished ophthalmoscopes as soon as possible to 46 Karlstrasse, Berlin. . . ."

Fig. 5 (Ullman). The most famous eye clinic of the 19th century. Shuttered and locked two days after the death and by the will of its founder. Never opened again. Torn down at the turn of the century.

Legends sprout up in the vicinity of the great the way bael fruit grows around the Kings of Kandy. It was said that on that day of jubilation and high holiday, the morning Helmholtz's instrument arrived, Graefe seemed to know by instinct how to employ it. He was soon using it to stare into the living orbs of Waldau, the somewhat apprehensive servants and in fact whoever came into the room that would submit to an examination, gazing spellbound, "silent . . . upon a peak in Darien."

It took longer for Waldau to get the hang of it. He tried for hours, and finally, when

hope was about to be abandoned, perceived the head of the optic nerve. Upon doing so, he yelled out, "Heaven be praised! tant mieux!" throwing the ophthalmoscope up in the air like a schoolboy his cap and kicking his heels in a wild caper. It hit the ceiling with a bang and knocked some plaster off, returning to bounce off the table.

Graefe managed to catch it before it landed on the floor, though how he ever did so, he never knew, he was laughing so hard. He refused to have the damage to the ceiling repaired, the story goes, but left it as a memorial to that gala day.

He began to work harder than he ever had in his life before, spending his mornings, often six hours at a stretch, in the clinic, his afternoons studying physiology, and his evenings after a postprandial call at the hospital, to apodictic investigations. Many a midnight he looked in upon his patients again before he went home for the night.

Graefe's scientific work "was not only work," as was said of Goethe. "It was also a prayer or confession." *To work is to worship* would have been a fitting motto over his door. Whether the thought ever came to him that he was a Messiah sent to reform ophthalmology is not known, but he performed even small and humble tasks as though they were sacred obligations. One job he took upon himself, no inconsiderable one for he sought exact terms and was not satisfied with less than precise descriptions, was to give a step-by-step account of everything he did, to the scientific world at large. He thought that to heal was the divinest function of man. When he wrote on medicine that was his way of preaching religion, and no St. Crysostom could beat him for fervor.

Two and a half years after he opened the Karlstrasse Clinic, Graefe had three matchless papers, compiled from a mountain of notes and records, ready for publication. They were entitled (1) "On the effect of the oblique eye muscles," (2) "About diplopia after squint operations," and (3) "About diphtheria of the conjunctivae and the use of caustics." In January, 1854, they came out in his *Archiv für Ophthalmologie*, a report 480 pages long, every word of which was penned by the young editor himself. In the foreword he wrote:

"Some of my colleagues might wonder that I, youthful and ignorant as I am, should make so bold as to found a periodical for ophthalmology, of which this is volume one, but I am convinced of the urgency of this step and have looked in vain for someone more qualified but with similar zeal to perform this needed function. None having come forward, I take it upon myself. . . ."

"Wer zu viel unternimmt, ist selten glücklich," Dr. Juengken's assistant, in anticipation of what was to come, said placatingly as he placed the heavy publication on his chief's desk. (He who undertakes too much, is seldom successful.)

The old surgeon began to turn the pages, at first slowly, then faster and faster. Finally he shut the book, his face very red. "I remember his father—yes, don't I!—with his noses and his Emperors! I remember this green sprig, too, at school here with his why-this and why-that and couldn't-it-be and wouldn't-you-say instead of holding his tongue and using his ears the way a scholar ought! And then nothing would do he must circumvent the globe—and then he must show how to run a hospital—," he broke off. "The drubbing he took a year or so ago would have satisfied any other man. But not him! Now he *publishes*, does he?"

"Money makes the mare go," his assistant murmered. "They say he inherited a great fortune. A rich man can always—"

"*Archiv für Ophthalmologie*, indeed!" Juengken said sharply. "Who does he think he is? Inform his masters, would he?"

"Dreams are not realities."

Another *Archiv* came out the following August, graced by Helmholtz's fine study, "Accommodation of the eye." Ferdinand Arlt and Frans Cornelis Donders acted as

co-editors. Until the year 1870, Graefe was to guide 16 volumes of the *Archiv* through the press. Herein would be published all his papers, a grand total of 2,500 printed pages, based on his own careful observations and knowledge, a treasure store the worth of which to ophthalmology is incalculable. Volume I cast fame's bright ray upon the 26-year-old compiler.

If the time would come when, sick and suffering, he was to write, as he confided in a letter dashed off at the time he was completing his last work, *On the Pathology of Glaucoma,* "more for my own solace than for others' edification," he had a very different purpose at the beginning. His intention then was about the same as St. Thomas Aquinas' when he commenced the *Summa Theologica,* he wanted to enlighten his fellow men.

Sometimes Graefe could not read his own handwriting, when it was worse than usual, and the habit of writing in bed would grow on him with the passage of years. He often said he intended to write a text book of ophthalmology someday, but not the way some did, propped up on all sides like a baby in a highchair. It would have to stand on his experiences, observations and learning, he said, as he did not expect ever to overcome his reluctance to "ask for help and beg other men to write whole chapters of a book about a subject I should either know from one end to the other myself or I shouldn't be writing a book about it in the first place!"

"I wonder," Donders said once whimsically, "I wonder if Pygmalion was as jealous of his Galatea as you are of this creation of yours . . . this ophthalmology. . . ." The enchiridion was never to be written.

Toward the close of his life he was working on a monograph about strabismus, on a "little book on eye muscle paralysis . . . which . . . begins to bore me painfully," and he had "planned and written in broad outlines" a book on "Surgery of the Eye." He wrote steadily until the very end.

But all this was to come.

In September, 1852, the time to which we now return, he had published very little. His treatise, however, "On the action of the ocular muscles," seemed to be of such significance that he was honored for it by an appointment to the medical faculty of the University of Berlin. Nothing at the time could have come as a greater surprise, and, a born teacher, as had been found during his days in Vienna, his delight was rapturous to say the least.

His mother was highly pleased also and, at a small family feast made to celebrate the event, she presented him with the watch fob given to his father by the old Emperor. Once more a Graefe would be an exemplar for the young at the University. Once more a Graefe would don professorial robes. They should be very becoming, too, she decided, putting her head on one side and surveying him.

About that, the young man was not much concerned; what he wanted was to do the job. If he had known then that he would get neither salary, patients, students (his course not being compulsory and no credits allowed for it) or equipment with his appointment, that it was without scope or standing, a dignification in name only, a kind of figment of the imagination, he would not have been so quick to rejoice.

At the beginning the new privatdocent had plans enough for half a dozen teachers. "The main thing is," he said, "I must not be dull. I must lecture in such a way as to keep everybody awake! even the young men who have been out all night. I must come to the point. I must make things clear. I must never mumble. In short, I must strive to make the human eye as interesting to the students as Gretchen was interesting to Faust!" he finished, full of excitement.

"That will be quite interesting," Waldau said, trying not to smile.

"And I must write up the paper on the symptom of eye muscle paralysis much more ambitiously than I intended at first—

make a book of it—something the school can be proud of! I'm going to use it as my habilitation thesis, you know. Do you know what I think I'll do? I think I'll write it in Latin!" He was as good as his word, beginning it that very evening and working until long past midnight.

The following morning he presented himself at the outer office of the Dean of Medicine. The dean's secretary, a thin man in epauletted black with a shaven dome like a monk's and a beard bushy enough to hide a setting of eggs in, had, it appeared, never heard the name of Albrecht von Graefe. He accepted without a flicker of interest the document of appointment which was handed him and disappeared somewhere behind a baize-covered door.

The professor-to-be sat back and looked around him at the familiar pictures on the walls. There was his father looking down at him! not smiling, grave, as behooved such a man in such a place, and yet, if one stared long enough into the clear wide-set eyes, they seemed to brighten and know one, the lips began to lift a little at the corners. . . . Now, what would he think of the ophthalmoscope, his son wondered, and what would he think of—

"Just what is the purpose of your visit, if I may inquire, Herr Doktor?"

Graefe looked around quickly, startled by the interruption. It was only the secretary who had come back without a sound. "I wanted to announce my availability for lectures," he said.

"I am sorry, Herr Doktor. We have no space for you to lecture in."

Graefe got to his feet. "Be so good as to announce my presence to the dean," he said stiffly.

"Yes, Herr Doktor."

Rudolph Virchow, 34 years old, sleek and plump, discreet as a lapwing, came forward to greet his guest with a click of the heels and a warm handshake. Such an honor! the young man who was performing miracles, was it? writer of that stirring paper on the ocular muscles! To what might he attribute the privilege . . . ? How could he serve . . . ? His heartiness changed to embarrassment as he listened to his visitor. There was some mistake, of course. Thousand apologies! So talented an acquisition to the faculty not to be given space to lecture in? There must be some misunderstanding. It was absurd. Professor Juengken was Professor of Surgery and Ophthalmology—he was the man to straighten out the tangle—chief surgeon—chair of surgery—no, there would be no purpose in going to see him—but he would clear up the matter. Yes, of course, his old professor—wasn't that interesting? Well, that made it all the better. The surgeon would soon make order out of chaos for an old pupil. "You shall receive a communication in the very near future!"

He made it sound so near that the young appointee would not have been surprised to find it on his desk when he reached home.

"I looked at him but he never quite returned my gaze," he said, telling Waldau of the episode a few hours later. "He kept looking just here, at my left earlobe. But he was very polite."

"What do you think it all means?"

"I don't know. Maybe nothing. But I had such a strange feeling."

"What kind of a strange feeling?"

"As if—as if somebody was close by there who would turn me to stone if he possibly could, the way the enchanters used to do in olden times—try to keep me from working—teaching—"

"But why? Who would want to do such a thing?"

"That's right, who would?" Graefe said, his face clearing. "I'm not going to worry about it. The Dean of Medicine said he would let me know. I suppose I'll be hearing tomorrow, or next day. . . ."

That night he took up his thesis again and continued to work on it until it was done. *The Symptoms of Eye Muscle Paralysis,* 175 pages long, written in rather halting Latin for the occasion of his receiving the appoint-

ment as privatdocent, was to be his only published book.

Weeks passed without word of any kind from the University.

Soon after the opening of the Karlstrasse hospital, Graefe was swamped with requests from students wanting to attend his clinic and hear his lectures. They came from England, Scotland, Ireland, Belgium and Holland, but no Germans were among them. Graefe had not posted his lectures. Now, however, he decided to place this announcement in the entrance of his establishment: "Clinic for Eye Diseases, A. von Graefe, Privatim, Mon., Wed., Sat., 10-12 a.m." Maybe, he thought hopefully, that would bring more than the strangers. Maybe, with apostolic faces, they would come for whom he had begun to long now with a longing like a cow's for her calf. Maybe that would bring his countrymen. . . .

Less than a week after the notice was posted, long after he had ceased to look for it, the letter from the University came. Not from the Dean of Medicine, signed by a name he did not know. "By the order of the Minister of Education, Herr von Raumer," it said, *"Privatdocenten who lecture outside* the University are forbidden to announce on the University blackboard their holding at the University any lectures or clinic unless they are authorized to do so by the state of Prussia."

"Oh, my hat," Graefe said, laughing derisively.

"They were just waiting to pounce," Waldau said, taking the communication and giving it a puzzled look.

Again the appointee called on the Dean of Medicine, but was unable to see him. Another visit brought the bland reply that the Dean was absent.

Baffled and angry, Graefe considered the situation. He and the university had come to a deadlock. He still had the appointment of privatdocent, but if he had no room in which to hold a class, he could not lecture; if he could not announce his whereabouts on the university blackboard—without a gold-sealed ordination by the state of Prussia!—he could expect to get no students. But what did it mean? Why should he be privileged to teach and at the same time be kept from teaching? No doubt there was a reason but he was too proud to try to find out what it was. He had paid three calls at the university and that was all he was going to do. He was a busy man. If they wanted him, they would have to come after him.

Eventually the difficulties, revealed for what they were and brought home to roost, would be smoothed out. An arrangement would be made. He could and would do certain work at the medical school. In 1857, the year of his sublimest accomplishment, when the Brussels International Congress of Ophthalmology stood up and cheered him, he would be appointed Extraordinary Professor at the University, but that, like the medal of the Order of the Golden Lion of Montenegro, was a mere ornament.

Nearly 16 years were to pass before the University might really be said to "want" him enough to give the chair of Ophthalmology and "come after" him to the extent that they made a special plea for him to accept it. And in 1852, 16 years, plus two, was all the time he had left in life.

For a long while Graefe, about to became one of the most famous physicians in the western world, was ignored by the Berlin faculty. A few German students, Josephs to his beaming eye, came to hear lectures but soon dropped out, and it was discovered that they had been threatened with credit losses for attempting to hear unauthorized lectures. Not being posted on the blackboard at the university, that was what his lectures were. And they were not posted because the Minister of Education had published an edict to the effect that a teacher who taught *outside* the university premises could not teach *inside* the sacred walls! This notorious bann was put into effect by whom? Graefe and his friends often pondered.

That year, Prof. Johannes Christian

Juengken (Papa Juengken) had been the Geheime Medizinalrath for surgery and ophthalmology at the Royal Charité for more than 12 years, and was the author of a book on eye operations. In it, he gave no credit to any of his predecessors. "Why should I?" he said.

If he was not old, he looked old, and his big heavy face was always turning as red as drachenblut. Could Madame Schopenhauer have seen him she would have thought he was an apparition out of her childhood. He did not wear a powdered wig, but his gray hair looked artificial and his mustache was waxed; no satins and ruffles, no gold-knobbed cane as high as the chin, but his pale green pantaloons fitted him like a second skin and his bugloss blue coat might have been tailored for an Actor Bergopzoomer. He wore a tall brown beaver hat, brilliant boots, white gloves, a carnation, a half-dollar sized stickpin, and on his forefinger was a large diamond ring he used not only for illumination—he would flash it in front of an eye he was operating on, in an attempt to make it reflect a beam of light where he wanted it—but also as a tool, to jab or press with, when necessary.

He had known Karl Ferdinand von Graefe and thought himself misprized by him; he remembered Albrecht not very flatteringly as a student who "asked questions."

Papa Juengken admired learning, experimentation and progress, but he often said that when the meat was roasted it ought to be taken off the spit! Except for a few minor details, almost everything had been learned, everything scrutinized, and if there was any place to progress to, which he doubted, he gave it out as his opinion that it would be found to be as bare as the moon.

He had heard all about the hospital on the Behrenstrasse and laughed about the advertisement in the papers. When the move was made to the Karlstrasse, he said, "Milord had to dig a bigger ditch for the beggars." Upon being informed that Graefe numbered the brother of the King of Würtemberg and a Duke's daughter among his patients, he shrugged his shoulders and said, "La Mode, that's all! Once it was stylish to wear a cod piece! But fashions come and fashions go!"

Mention of Graefe's successful operations came to his ears. "I would like to have a pfennig for every *quid pro quo*," he said. "I'd be a rich man." Reports of his former student's venturings and his triumphs in the field of ophthalmology bored and irritated him. "Whom did His Gracious Majesty call on when he needed a surgeon?"

The year before, in 1850, when chloroform was tried out in Germany, Frederich Wilhelm IV, who had bequeathed the zoo to Berlin and always took a proprietory interest in it, suggested that the zoo's bear, recently blinded by cataract, be used for the experiment. His Majesty wondered if his sight couldn't be restored while under the influence of the anesthetic. Professor Juengken, a picture of sartorial splendour, his diamond glittering, performed a perfect couching operation. Whether or not it did poor Bruin any good, however, could never be ascertained for the reason that he slept right on into eternity.

His colleagues were not surprised to hear of the injunction put out by von Raumer. Whether or not the obstinate old surgeon had a finger in the pie could not be sworn to but it was known that he and the Minister of Education were on a very friendly footing. When word was spread as to who had been appointed privatdocent, certain gownsmen wondered how soon the young man and the fire-breather were going to lock horns. There was considerable disappointment when the education minister stepped in with his proclamation, and the two never got together at all. The battle never came off, but that it would have been hot and heavy, no one doubted.

It was said that Papa Juengken turned as blue as Chinese wistaria when he heard that Graefe had opined, "Surgery and ophthalmology are two different fields. Surgery

needs one kind of preparation, training, and technique, while ophthalmology needs another. They do not belong together!"

"Don't they?" the old man roared to the frightened tattler, his long-suffering assistant. "And who says they don't, will you tell me? A young whippersnapper who would drown if he stood outside in a hard rainstorm for the reason that his nose is turned up so high! On what authority? By the say-so of a perfumed Frenchman and an overbearing Englishman! Now, listen to me," he said, "ophthalmology will always be a part of surgery, and a small part at that. To try to aggrandize it, to try to make it stand alone, is like trying to make my leg here go wandering away by itself down the Unter der Linden!"

In the days to come, he was often to repeat the pious admonition, "Ophthalmology belongs to surgery, and the proper man to teach surgery is the surgeon."

Some 50 years previous to this time, in 1801 to be exact, Professor Himly of Göttingen made the rash public statement that eye surgery and general surgery were two different propositions altogether and ought to be delivered one from another. Professor of Surgery and Ophthalmology at the University of Göttingen, he was the discoverer of the mydriatic effect of atropine and had just brought out the first number of the only magazine up until that time ever to be devoted solely to ophthalmology, the *Ophthalmologische Bibliothek*. It came out that once, and twice more, and then failed.

A man named Titternich in Leipzig pleaded for separation. Van Ammon wrote a thesis on the premise that cataract operations should be forbidden to surgeons. But the wilderness stretched too wide for their chaffinch chirpings.

In England there were some mild and even pointed objections, but the dogma prevailed that God had intended the surgeon to operate on eyes! But by midcentury a change was evident. Bowman, who used to say "I am a surgeon," coldly, when anybody referred to him as an ophthalmologist, now let the term pass uncontested.

Light had drawned in Vienna and Prague, where Jaeger, Sr., in the one, and Arlt in the other, occupied chairs of ophthalmology at the Universities. Elsewhere, however, in the year, 1852, darkness reigned and antagonists "clashed by night," until those three arose, planetlike, at almost the same hour, to shine steadily and clearly from that day to this—bright, brighter and brightest—Donders, the physiologist, Helmholtz, the physicist, and Graefe, the physician. Their spheres stood apart, but all had the same intention: to understand the process of seeing, improve vision, alleviate pain and diagnose and cure eye diseases.

Every university is a hotbed of gossip, and another rumor went round the autumn that Graefe was made privatdocent. The rumor was that more faculty members than just the Geheime Medizinalrath had reason to be jealous. It is not to be wondered at. For if the very Creator Himself was supposed to have made the statement, *I am a jealous God*, how could anybody blame, for instance, the Dean of Medicine, if he happened to feel the same way? It took a very broadminded protagonist to countenance a second star performer in his show! But the time would come when the ophthalmologist and the founder of cellular pathology were to be the best of friends.

Circumstances one day obliged Graefe to pen the following letter to the Minister of Education, a post then held by Herr von Bethman-Hollweg. On the 17th day of March, 1859, he wrote:

". . . Eight years ago, returning from long travels begun and pursued for the study of ophthalmology, I established a clinic here in Berlin, as you no doubt are aware, and have been very fortunate in my endeavor. . . . Many patients and visiting foreign physicians have made it necessary for me to maintain a large staff of assistants, teachers, and other help. My clinic now has 120 beds which take care of some 1,400 in patients a

year. The out-patient clinic sees 6,000 new patients a year and a total of 50,000 patients. In the last two years 1,600 major operations were performed. During 1858, 300 registered student-physicians studied with us here at what I believe has become the largest eye clinic on the continent of Europe, second only to Moorfield's Hospital in London, where six surgeons work. I do not mean to boast when I say I think we beat them in the way of patient material and the type of operations performed.

"In spite, however, of the size of our institution, it is not big enough. So many patients and students are now appearing that we cannot accommodate them all. I considered branching out still more but reflected that the exercise of scientific research, literary endeavors, and my teaching activities would suffer if I did so.

"Also, the responsibility of a huge personnel dependent upon me alone for place and subsistence scares me. I have no more of a lease on life than anybody else, and what if they should all be thrown out of work by my demise? Too, I am inconvenienced from time to time by the loss of my best assistants, who rightly accept positions of authority with high pay when they have a chance, with other organizations. Numerous foreign clinics are now being conducted by men who have been my élèves. Thus, I am in the process of losing Dr. Liebreich, a very fine ophthalmologist, who is going away to superintend an eye clinic in Riga.

"These considerations . . . have forced me to conclude not to expand even to the extent of adding one more bed or hiring one more helper. A private individual of ordinary fortune can go just so far and no farther. I feel I have reached that point. . . .

"I do not consider abandoning my hospital as it stands, although if I were offered a chance to teach and do research in a public institution especially designed for the development of the new science of ophthalmology, I would be greatly tempted. It is my humble opinion that such an institution, endowed by the state, could be made to pay its own way in a few short years. The good it would do—and the need for it—is incalculable.

"But the purpose of my letter . . . is to beg your excellency to allow me, if you can do so, some beds in the Charité for the overmeasure of patients who come from far away, to get our help, especially in the spring months. It goes without saying I would admit to the Charité, should I have the privilege of using its facilities, only such patients as are able to pay the customary rates, to the interest of the hospital, I should imagine. I am, dear Sir, your Excellency's most humble obedient servant. . . ."

Two weeks later Graefe was appointed director of the eye clinic in the Charité, not by any means the triumph it appeared at first sight. Von Raumer's edict still being in effect, he was not permitted to teach. Papa Juengken would continue to lecture as he saw fit on ophthalmology at the university for another decade and then, an ancient man, totter off to outlive his "rival" in pensioned retirement.

The mandate that had embarrassed and hindered Germany's greatest ophthalmologist so long would be nullified that same year. . . . Some of the faculty thought Graefe's lectures were "too high, too complicated and unintelligible for the students at the University and were devised to please foreign graduates." After his long wait, there were to be few of these controversial lectures as he conducted university classes not much longer than a year.

His students were enthralled. Whenever possible, other classes and lectures were given over as poor pale stuff beside what he had to offer. He was everything a teacher ought to be. Once he confided to his friend Horner that these university audiences and the long line of other students he had lectured to, seemed to prove that the stock was getting better all the time!

"For never in the world before," he said, "were there such receptive, intelligent and fervid young men."

"That may be," Horner murmured, "but I wonder if the sun has any idea who's supplying the neighborhood with all the fine moonlight?"

On January 3, 1869, in a letter to Donders, Graefe said, "Today I have been thinking of the words Helmholtz sent to cheer me long ago, 'Ophthalmology is for medicine what astronomy is for physics: the Model,' and how they once lifted my low spirits. I cannot imagine what words would do so this wintry evening. 'Death is short and life is long'? Ah, yes, perhaps . . . but none is by to play the hypocrite. Yesterday I sent in my resignation as Professor of Ophthalmology at the Charité. In the end, my lungs turn traitor and establish a prohibition as stern as old von Raumer's was. . . . And who has the last laugh, I do not know. . . ."

But we have got ahead of our story. Let us return to the happy years from 1851 to 1857 when he had his biggest fish to fry. He was always coming up with some novel scheme in those days, always experimenting with new ways of doing this or that. His assistants said they were almost afraid to see him arrive, invariably late, at the clinic in the morning, for fear of what he might have up his sleeve! Not that they minded very much. For example, to be instructed as to the advantages of studying the cornea by focal illumination, using light through an ordinary magnifying glass. They soon saw the sense of that, and so did a good many others, one consequence of which was, that diseases of the cornea began to be recognized right and left.

Not long after, it occurred to Graefe that, if he wanted, in a case of squint, to determine the function of an isolated muscle, he would have to disassociate the images of the two eyes. For this purpose he held a red or purple glass before the paralyzed eye, which simple device enabled him to classify all types of eye muscle paralysis. With classification came an understanding of muscle function never before possible, which in turn led to new methods of operating squint, and to the conception of amblyopia and the picture of noncorresponding retinas.

There was more and more to come, and little did the sightless beggar, arrested in his rags for knavery—pretending to be blind when all the time he could see as well as you or I! reading a newspaper, he was, *in flagrante delicto*—little did he imagine what an inspiration his goggle-eyes were to be to the great eye doctor before whom he was dragged for final judgment:

"The Magistrate requests the gracious Herr Doktor von Graefe to decide whether this man is blind or not. He lives as a blind beggar and begs from people on the street. But all the time he can see!"

"How do you know?" Graefe asked the police escort who had spoken, the larger of the two uniformed men. He put a kind hand on the wretch's trembling shoulder. "Come here, my friend. Just sit here on this stool by the window." He noted how the man tried to obey, feeling nervously ahead of him, the way he bumped into the corner of the table, the oddly stiff manner in which he turned his face from side to side. He helped him to his perch. "Don't be afraid," he said. "Now, tell me. How do you know?" he repeated over his shoulder.

"Why, Herr Doktor, because he was sitting in a doorway *reading*, that's why! and a blind man who can *read* is no blind man, your graciousness must surely know!"

Graefe made a brief examination. "This is one case where a blind man who can read is nevertheless a blind man," he announced.

"But, Herr Doktor, how can that be?"

Ignoring the policeman, he spoke to Waldau and Baenziger his assistants, standing by and watching with great interest. "His visual field is contracted to the size of a little circle of light no bigger than a gun bore," he said.

"In fact, what seeing he does is like looking out through a gun barrel—a long tunnel of darkness with a little aperture of light down at the end."

"Yes, yes," the man said excitedly. "It is exactly like that!" He began to weep with joy. "Sometimes I pick up an old newspaper and I can see one word after another through the little hole, but it takes a long time and when I lose my place I have a hard time to find it."

"Come back soon and we will see if we can make life a little less difficult for you," Graefe said to him. "He is blind enough, poor soul," he added to the bailiff. "May you nor yours never be the same." Then he took up the ophthalmoscope and looked into the beggar's eyes seeing and recording what is known today as *retinitis pigmentosa.*

Teased by the remembrance of the affair afterwards, the idea came to Graefe that sight was composed of central as well as what he decided to call eccentric vision, and he projected to study it a new way, from a new angle. He could not begin at once, however, needing equipment not then in existence, but after some experimenting he produced a crude but expedient contrivance, using the principle of perimetry, that he thought would do.

By systematic examinations of poor vision, a routine in his clinic from then on, with this new tool and with the ophthalmoscope he was able in the years to come, to discover, describe and establish once and for all the clinical pictures of atrophy of the optic nerve, hemianopsia in its various forms, detachment of the retina, embolism and thrombosis of the central blood vessels, the choked disc, and the excavation of the disc in glaucoma. He found a cause for the former in the rise of pressure within the cranium and ditto for the concavity of the latter in the rise of tension within the eye.

From the beginning of his practice he had been haunted by certain blind eyes that appeared, those with a maximally dilated pupil shining with an eerie greenish light. They had been described by the Greeks, but it was never recorded that anyone had discovered, as Graefe soon did, that these eyes were hard, with a hardness varying in degree from that of a gem to a soft rubber ball, but all harder than the normal eye.

The hardness became an omen of disaster, he perceived, when the artery at the head of the optic nerve could be seen pulsating, also when the disc showed concavity, and the third danger signal was when the visual field became contracted. Any of these presaged blindness to the eye, and careful observation backed up his hunch that all three symptoms had the same underlying causes: increased intraocular pressure.

The Greeks named the blind eye, end-product of disease and no longer subject to cure, *glaucoma,* "green eye," but that was as far as they went, and that was as far as any physician or school of physicians in any age or culture went, until Graefe came upon the scene.

He had the ophthalmoscope, true, which no living man had had before him, but which dozens of his contemporaries possessed, he had Donders' primitive and unreliable ophthalmotonometer, soon to be laid by in favor of his own two forefingers, and the kindly Hollander's physiologic counsel. He was equipped, therefore.

And he was a wise man, with wisdom's usual intuition and patience, perception, comprehension, ability to assort and to utilize. But he had and was something more, what initiates of Osiris in antique times might have called by that title higher than a King's—Child of Earth and Starry Heaven. Being which, he had a muse, under whose gently moving wings his powers caught fire and blazed into the lambent flame of genius.

To discover what glaucoma really was, was to speculate how to relieve it. At first he tried derivative methods such as laxatives, leeches, and blood lettings, all to no avail:

"Then I decided I must concentrate on the eye itself," he wrote. "I tried mydriatics, which was calamitous. Next . . . having ob-

served that the steadily rising tension in an acute glaucoma led inevitably to amaurosis, in a matter of hours or days, I concluded that the tension must be decreased by some means. In the first cases I used paracentesis of the anterior chamber, which established for me beyond question the interrelation between glaucomatous symptoms and high tension. But the victory was short-lived.

"I asked myself, how can I obtain a lasting change in pressure? The effect of iridectomy . . . in chronic iritis was, of course, familiar to me, particularly when scar formation was the reason for the operation. I knew that whenever I had performed an iridectomy in a case of partial corneal staphyloma, the eye became and remained soft thereafter. . . ."

His first case was that of a 51-year-old woman. "My first cases . . . which might not have been selected fittingly enough, left room for doubt. My technique was also to blame, but as it and my skill improved, so did my results. I began this method in June of last year and have used it in every case of acute glaucoma since."

In a period of six years he was to perform 400 iridectomies for glaucoma.

"The immediate results of those earliest cases were startlingly good, but recalling the disappointment that followed the first elation I felt after doing a paracentesis and seeing the tension fall only to rise again in a few hours, I was suspicious, and I continued to be suspicious until time proved that a lasting cure had been effected. It is now 14 months since that first attempt; it and a large number of cases have been under observation for more than nine months. That the iridectomy is the true remedy against the glaucomatous process I am absolutely convinced . . . of course, within the limits of human capacity. . . ."

It might be interesting to insert here a letter written in 1877 by Professor Arlt of Prague:

"In February, 1857 . . . Graefe wrote me about his successes with iridectomy in glaucoma. At Easter time I hastened to Berlin, for you know seeing is believing. The operation . . . and subsequent relief . . . was the greatest surprise of my entire experience. I became even more of an admirer and ardent follower of my former student (those years of the past seem like a dream, when he was a boy—and such a boy!) after seeing this near-miracle. Ten years later . . . I found myself in the all but enviable position of having to perform Graefe's operation on my poor wife's left eye . . . and by it I saved her eyesight for the remaining decade of her life. . . ."

Albrecht von Graefe was in correspondence with most of the significant ophthalmologists of his time, in Europe and America. He picked 15 of them, among whom was Prof. Friedrich Horner of Zürich, a great favorite, devotees to ophthalmology like himself, and sent them invitations to meet with him in Heidelberg "to discuss problems of ophthalmology."

They all accepted, and after three days of informal discussion, the group moved on by steamship up the Rhine, enjoying the mountains, vineyards, castles, beautiful fields, and pretty villas scattered among the trees as these and other sights flowed away to either side of the dimpled river.

To Brussels they went where the first International Congress of Ophthalmologists was about to begin. The month was September, the year 1857, and here Graefe presented in French his famous paper, "Iridectomy and glaucoma," for which he received an ovation that astonished him. His audience stood up and cheered until the rafters rang.

When the Congress had adjourned, Hess and Horner went home with Graefe and helped him to get his paper ready for publication in German. Frau Graefe lived to know her son as famous as she had hoped, but two months later, on a drear day in November, he "kissed her good little hands . . . and gazed for the last time on the face of the best and most understanding mother ever created upon earth. . . ."

He was never to abandon the problem

of glaucoma. His last great paper "On the pathogenesis and cure of glaucoma" filled the 15th volume of the *Archiv* in 1869. It was his swan song and this was the last *Archiv* to which he contributed.

When the Heidelberg Ophthalmological Society was organized in 1863, Graefe's 15 chosen friends and colleagues were the nucleus of it. This society is still active and, before World War II, had over a thousand members, half of them non-Germans. He belonged to many other societies either as an honorary member or taking an active part. The Society for Scientific Medicine, founded in Berlin in 1844, was converted to the Berlin Medical Society in 1858, the year after the Brussels Congress, and it was Graefe who was elected its first president, an office he was to hold until his death.

In 1860, his very important paper "On sympathetic ophthalmia" appeared, in which the mystifying phenomenon of an injured eye causing in its fellow a sympathetic iritis so severe that destruction looms, unless the culprit eye is removed, was described for for the first time.

And now comes a little tale. . . . According to his Belgian pupil, Warlemont, it was at the second meeting of the German Ophthalmological Society in September, 1864, that Graefe listened to a paper given by George Critchett of London on "The extraction of hard cataracts after the performance of an iridectomy."

At first Graefe listened gravely, said Warlemont, but then he began to be amused. It was all he could do to keep a faint smile from playing on his lips. For here was his own long-lost child, abandoned as ineffective some five years before, uncovered, adopted and sponsored by a foster father who had no idea that the offspring was not his own! Graefe did not spoil his fun, and in fact was to feel grateful to him later for re-introducing this old belief and method to his mind and thereby engendering a new interest in cataract extractions.

Julius Hirschberg tells the story in his history of ophthalmology, but he puts forward the claim that Julius Jacobsen was the man who read the paper that inspired the master! (Thus do they twine around the great the legends.)

From this meeting, Graefe went with Zehender, Hess, Horner, Baenziger and A. Weber to Varenna, on Lake Como. They were sitting out on an open veranda overlooking the water, talking and laughing; it was midnight, the bright white moonlight shone down, at least so history tells us, and in this lovely setting, among this jolly company, Graefe posed a sudden arresting question. "How would you describe a linear incision on an eye?" he asked.

Procuring a pencil and paper, he took a seat in the shaft of lamp light streaming through the open doorway, propped the paper on his knee, and began to sketch a knife. He said he was going to have it made when he got home, and he said a great deal more. He had been thinking of it, he explained, ever since that paper on the extraction of hard cataracts was read. . . .

When he finished the drawing, they all crowded into the brightly illuminated salon to look at it. It looked quite ordinary but it would, he said, be superior to every other known cataract knife for the following reasons:

It would connect the point of entry with the point of exit by the shortest possible route; the fact that the blade would gradually thicken towards the handle would prevent the loss of chamber water as the knife proceeded into the eye; and the cut made by the knife would be placed exactly in the limbus. He demonstrated the movements he intended to make so vividly that the gathering seemed to see the knife in his hand, the first incision made.

"You are no doubt wondering how I am going to get this knife out of the eye now that I have got it in there," he said smilingly. "This is what I propose. . . . The initial cut, as you can see, is a linear one. All I have

to do is to lift, lift the handle and go forward, to make a nice semicircular cut big enough to deliver the lens *but releasing the chamber water before the cut is completed* and defeating my purpose, because the tissues will be traumatized and the chances are that the cut will not be in the limbus. Therefore . . . my intention is to try to get around the sudden loss of aqueous by using the knife like a saw—always, of course, pressing upward. And here is what would happen.

"We are so far in that the point of the knife sticks out on the opposite side—we begin sawing upward—once—twice—no cessation of motion, mind—three times—up, up—and we are out! No premature collapse of the eye, the least damage to the tissues, and plenty of room to deliver."

He looked around, his blue eyes sparkling.

"Well, gentlemen, how does it sound to you? What do you say?"

"*I* say," Horner said, scratching his head, "I say that I think this calls for a little champagne! I don't *know*, of course, and none of us know as yet, but that's what I *think*, that this is a time for rejoicing."

August Lutter, instrument maker and bandagist to the King of Prussia, the man who supplied Graefe with his instruments, made the knife and it proved equal to his and to the every hope of all his loyal friends. Nearly a century later, it is still in use. The operation itself goes by different names—peripheral linear extraction, as the English and Americans call it, modified linear extraction, as Graefe called it in 1865 and as the Germans call it today, and the "grand circle of the orb," as some Frenchmen romantically call it.

"I am sending you a set of instruments I now use in my linear extraction," he wrote a few months later to Donders, who, by the way, never learned to be a really good surgeon, although he came to Berlin every year as regularly as the spring rolled around, to take a set of "lessons" in the art of surgery from Graefe or his most talented assistants. "I no longer employ a spoon, as I told you, but always use my new knife. To open the capsule I use a special little hook. . . ."

He also wrote soon after: "In cataract extractions I have abandoned all traction maneuvers in favor of tumbling the lens."

By this mention of a method used by many ophthalmologists today in preference to other forms of extraction, it may be seen that he never rested but was constantly striving to improve his technique and skills.

In 1864 Graefe described the eye symptoms of toxic goiter, then called Basedow's disease, the most conspicuous of which is the fact that when the eye looks down, the lid does not follow, an observation familiar to the student as Graefe's sign.

*(To be continued)*

### Ophthalmic Miniature

"We see, like those who have imperfect sight,
The things," he said, "that distant are from us."

Dante's *Divine Comedy*,
Inferno X, 100.

# NOTES, CASES, INSTRUMENTS

## CYCLODIALYSIS WITH GONIOTOMY IN CONGENITAL GLAUCOMA

PAUL L. CUSICK, M.D.
*Detroit, Michigan*

In any article on congenital glaucoma, one must first pay tribute to the numerous contributions of Barkan. He has established goniotomy as the treatment of choice in infants with corneas of 13 mm. or less in the horizontal diameter. His most recently published article[1] contains an excellent bibliography to which the reader is referred.

Cyclodialysis alone, in most hands, appears not to have been as successful as goniotomy. The basis for the success of goniotomy, as done by Barkan, seems to be the exposure of the trabecular area by stripping of persistent mesodermal tissue. The root of the iris recedes with this procedure.

The percentage of success in this condition with any procedure appears much higher when an early diagnosis has been made.

Goniotomy as done by Barkan requires that the goniotomy knife be carried across the center of the pupillary area and then into the angle where, under observation with the contact lens, the "stripping" is accomplished. This requires an experienced assistant who assists by rotating the globe. Scheie,[2] does the operation without the contact glass.

The average ophthalmic surgeon does not see a large enough number of these cases to become expert in Barkan's technique. Among the difficulties encountered are:

1. Loss of the aqeuous with the knife blade across the pupillary aperture.. The lens may be injured on its withdrawal.

2. If the point of the knife is slightly posterior, the iris may be torn, resulting in hyphema.

3. Difficulty in keeping a perfect arc in rotating the knife and the eye.

4. Difficulty in visualizing the trabecular area due to ruptures of Descemet's membrane and edema.

Various methods to avoid some of these difficulties have been devised, the most recent being Troncoso's[3] but it still remains a tedious procedure for most of us.

### TECHNIQUE

Over a three-year period I have used a procedure which appears to produce results comparable to goniotomy and which can be done by any experienced ophthalmic surgeon. This combines a cyclodialysis with a "scraping" of the trabecular area (deVincentis[4]).

The instrument* used consists of a cyclodialysis spatula surmounted at the toe by a wedged elevation, three mm. in length, the posterior edge, or base, of which is sharp (fig. 1).

The pupil is made miotic and a preformed channel for saline is made, its size is exaggerated in the artist's drawing (fig. 2). The conjunctiva and Tenon's capsule are

* The instrument described is made by Storz, Saint Louis, Missouri.

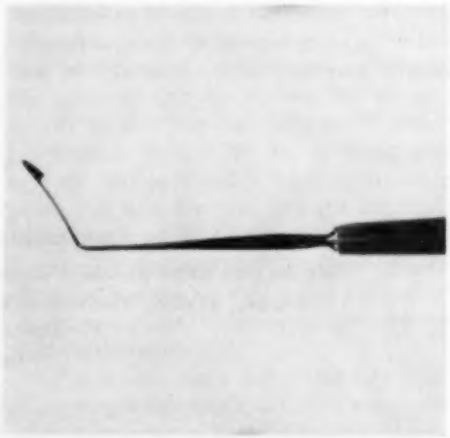

Fig. 1 (Cusick). The instrument used in cyclodialysis with goniotomy.

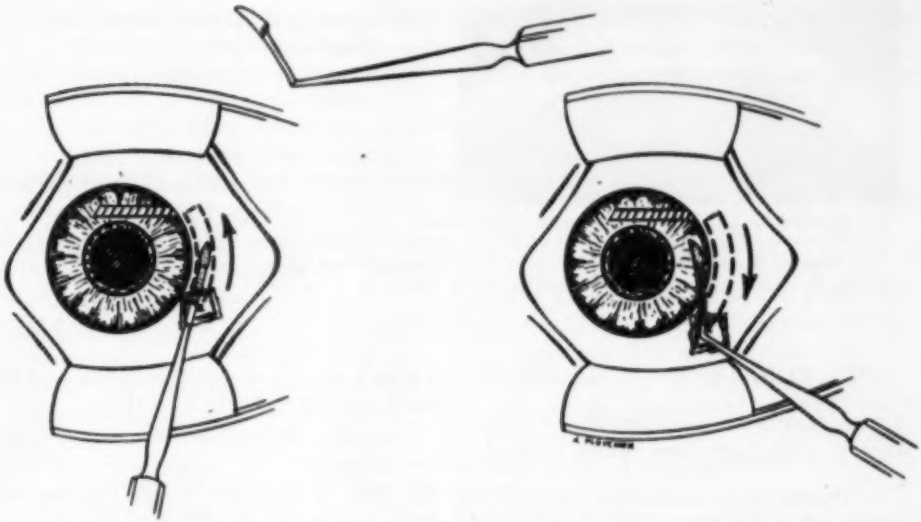

Fig. 2 (Cusick). Technique of cyclodialysis with goniotomy for congenital glaucoma.

incised and a meridional incision is made in the sclera four to five mm. in length and two to three mm. from the limbus (fig. 2).

The toe of the instrument is inserted, being held tightly against the sclera and a pushing type of cyclodialysis by the Iowa method[5, 6] is accomplished, attention being given to separating the iris root rather than employing a sweeping motion.

The anterior edge of the incision is elevated at the time of introduction by a suture or corneal forceps. When this has been accomplished, the toe is moved into the anterior chamber, slightly, and with the blade held against the angle as it is withdrawn, the trabecular area is abraded. The blade is then rotated or reinserted and the same procedure carried out over the upper temporal angle.

The scleral incision is then closed together with the conjunctiva, saline is inserted through the preformed channel to reform the anterior chamber. Pilocarpine (two percent) is instilled postoperatively.

## Results

Eleven eyes have been operated. Seven could be considered good cases with corneal diameters of less than 13.5 mm., and the results were excellent.

In one case there was an associated nevus flammeus. Barkan[1] mentions two such cases responding to goniotomy. Previous iridencleisis in this case had been unsuccessful, and severe intraocular hemorrhage had occurred in the left eye, which was much enlarged from birth. Cyclodialysis with goniotomy has controlled the tension in the remaining eye over a two-year period. Recent examination reveals a tension of 22 mm. Hg (Schiøtz).

Three cases with much enlarged corneas were not benefited by this procedure and cyclodiathermy was used to lower the tension.

Gonioscopically the appearance postoperatively in a successful case reveals that the iris has receded and the angle is deeper than on the unoperated side. Several areas of iris atrophy are usually seen, with multiple small clefts in the ciliary area. At the upper and lower terminus of the operated site an anterior synechia is usually present, suggesting that an attachment from the anterior iris has been removed.

The area adjacent to the incision may be

thinned and appear quite dark through the conjunctiva. For this reason it is suggested that the incision be made well up in the superior temporal quadrant where the lid will cover the area postoperatively.

*28 West Adams Avenue (26).*


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## TESTING INFANTS' VISION

### AN APPARATUS FOR ESTIMATING THE VISUAL ACUITY OF INFANTS AND YOUNG CHILDREN


BLAND H. SCHWARTING, CAPT. U.S.A.F. (MC)
*Dayton, Ohio*


The estimation of the visual acuity of a child by this homemade device takes particular advantage of the early visual reflexes as well as the natural curiosity of a child. When in darkness, even a small baby will direct its gaze toward the sudden appearance of an illuminated field provided it is not bright and glaring.

This field is obtained by a frosted-glass window incorporated in a box containing a light bulb (fig. 1). The light is controlled by a switch behind the box. With the infant's eyes directed toward the illuminated field, an object moving across the field, provided it is visible and not moving too fast, will usually elicit the "following reflex." This can be noted objectively by the examiner by noting the synchronous movement of the eyes.

With this instrument, the moving objects are steel wires of various sizes in the form of small wands which are fitted on the swinging arm of a metronome. The wands are weighted so that there is a uniform speed of motion with each wand, which in this case is 40 half-cycles per minute. An ordinary rubber band around the metronome near the bottom of the swinging arm can be used as a start-and-stop mechanism by means of an attached string controlled from the rear of the box (fig. 2).

The vision is tested at one meter with the box at eye level. Most of the smaller children do better in the mother's lap. The smallest wire which can be followed by the eye (eyes) is the index to the visual acuity. Searching movements are ignored and only a smooth, synchronous response for at least one-half cycle is taken as a positive reading.

The testing is done in the dark (fig. 3) and, as soon as a positive response for one wire is elicited, the box is immediately turned off and room lights turned on while the wand is changed. With short exposures and variations of starting or stopping the metronome before or after the box is turned on the attention can be maintained surprisingly.

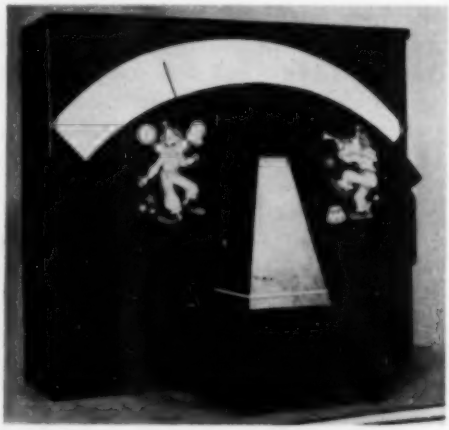

Fig. 1 (Schwarting). Front view completely assembled.

If a child cannot fix a light, the vision is too poor to measure with this instrument.

These targets were calibrated as to Snellen visual acuity by testing many adults with varying visions by both the Snellen chart and with this instrument. The correlation was found to be so consistent that the Snellen visual acuity could be predicted from results of testing with this device. The caliber of the wires with the approximate visual acuity equivalent follows:

| Diameter | Snellen |
|---|---|
| 20 mm. disc | Finger counting |
| 1.5 mm. | 20/400 |
| 0.75 mm. | 20/200 |
| 0.35 mm. | 20/100 |
| 0.15 mm. | 20/50 |

It is frequently impossible to check a baby's vision one eye at a time. In cases of monocular squint, the child is checked binocularly and the visual acuity thus obtained is attributed to the fixing eye. The visual acuity of the other eye may be obtained on follow-up visits when the fixing eye is being occluded. In other cases it can be checked after the parents have obtained acception of occlusion of one eye or the other at home.

An example of the usefulness of this gadget is cited in the case of a nine-month-old baby with bilateral congenital glaucoma. Both eyes had an elevated tension under deep anesthesia. The right eye had had recurrent episodes of corneal edema and its disc showed questionable cupping. Following a bilateral goniotomy-goniopuncture both corneas remained clear and the child asymptomatic.

Fig. 2 (Schwarting). Demonstration of start-and-stop mechanism with wand attached.

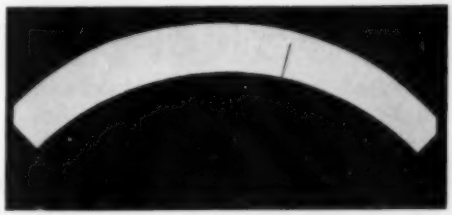

Fig. 3 (Schwarting). Patient's view under test conditions.

Checking each eye separately it was noted that each eye could fix a light; however, when checked on this device it was found that the visual acuity of the right eye was approximately finger counting while the left eye could follow the 20/200 wire, a normal vision for the baby's age.

A series was also run on normal infants and children with the following results which are believed to approximate the normal visual acuity at the various ages:

| | |
|---|---|
| 3 months | Finger counting |
| 6 months | 20/400 |
| 1 year | 20/200 |
| 2 years | 20/100 |
| 3 years | 20/50 |

It is important that the child be reasonably corrected, refractionally speaking, when testing, and of course should not be atropinized.

When attempting to determine the visual acuity of an infant it is felt that the basic nature of infants in general should be relied upon rather than some of the more varied acquired reflexes. In this way it is believed that this instrument can elicit this information which is otherwise so persistently withheld inside of the infant's own world.

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# A RARE MALFORMATION OF THE EYES*

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*San Cristóbal, Táchira, Venezuela*

The most important malformations and developmental defects of the entire eyeball are anophthalmos, congenital cystic eye, and microphthalmos. These conditions are well known and have been adequately described in the literature. The case to be reported does not quite fit into the types of malformation just mentioned, so that we consider its publication justified.

## CASE REPORT

*History.* A seven-year-old boy was admitted into the hospital for a malformation of both eyes, which had rendered vision impossible and had been present since birth. He had been living in a small village and had come with his parents to the town in order to beg alms.

The patient was the youngest of 16 children of healthy parents. His brothers and sisters were all in good health. The mother of patient was in excellent health during pregnancy.

*Clinical examination* showed a pale, somewhat thin boy. Apart from the eyes there were no malformations. The X-ray picture of the skull was normal. No lesions were found in the organs of the chest and of the abdomen. Examination of the ears and nose and throat was negative. There was a slight hypochromic anemia, slight leukocytosis (12,000 p.cm.³), and an eosinophilia of 19 percent. Stool and urine were negative.

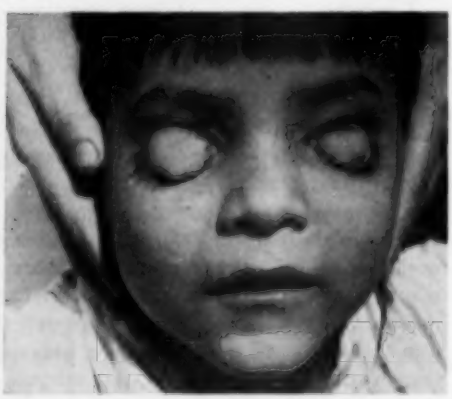

Fig. 1 (Morales and Hartz). Appearance of patient.

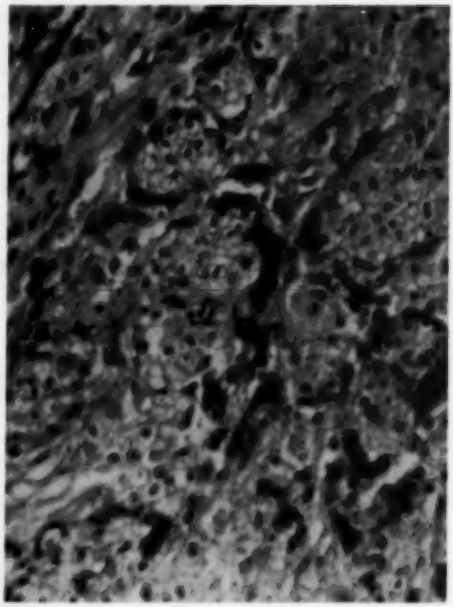

Fig. 2 (Morales and Hartz). Choroidlike tissue (×175).

*Examination of the eyes* showed, protruding between the eyelids where the eyeball should be, two small patches of skin. The eyelashes, which had been pushed apart by the patches of skin, and the eyebrows were intact. The skin composing the patches had a normal structure and was covered by fine hairs; it continued directly into the inner side of the eyelids without the formation of a recessus. There was a narrow zone in which the skin was light-red, thus forming a zone of transition to the conjunctiva of the eyelids. In the inner third part of each patch, there was a small depression about five mm. deep.

It was decided to explore the orbital cavity and accordingly one of the patches of skin was removed under general anesthesia. The orbit was filled with adipose tissue through which a small, firm globe could be palpated. This was dissected from the surrounding fatty tissue and partially extirpated. The small globe looked like a small eyeball without anterior segment; the inner lining appeared to contain pigment.

## MICROSCOPIC EXAMINATION

*Skin.* The epidermis was thin and showed clearly a stratum corneum and a stratum granulosum. There were many rudimentary

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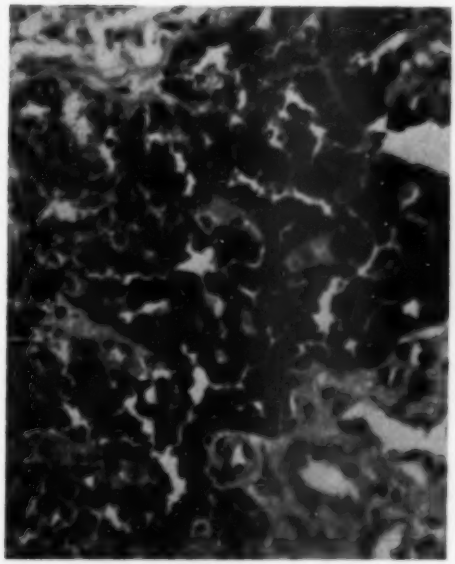

Fig. 3 (Morales and Hartz). Pigmented epithelium (×350).

hairs without sebaceous glands. In the subcutaneous fatty tissue, there were sweat glands, exocrine in type. At one extremity of the piece of skin, there were structures of another nature: striated muscle fibers, sebaceous glands without hairs, and sweat glands of the apocrine type.

There were also a few glandular structures of the serous type, resembling lacrimal glands.

*Biopsy of the eyeball.* The wall of the small globe showed three distinct layers and a fourth type of tissue occupied part of its lumen. The outer layer consisted of hyaline connective tissue with only a small number of fibrocytes and few blood vessels. The middle layer showed a more complex structure and consisted of bundles of nerve fibers of which a few were myelinated but the majority amyelinated. Between these bundles there were many pigment cells, usually of the spindle type, and a few large cells, with a large pale nucleus and a conspicuous nucleolus, which must be considered to be nerve cells. There were also unstriated muscle fibers. The inner part of this layer contained many wide, thin-walled veins and wide arteries with thin media.

The innermost layer of the wall of the small eyeball consisted of a mass of closely packed pigmented cells. Only the form of these cells which contained little pigment could be distinguished with exactness; the majority were cubic or, more rarely, cylindric. The nuclei of these cells, which resembled epithelium, were oval and contained little chromatin.

The tissue found in the lumen of the eyeball consisted of elongated glial cells and fibers. There were also small foci of calcification. Inside this glial tissue there were rounded masses of hyaline connective tissue which contained blood vessels. In one place we observed contact between the middle layer of the wall of the eyeball and tissue in the lumen through an opening in the pigmented epithelium.

No lens epithelium was observed.

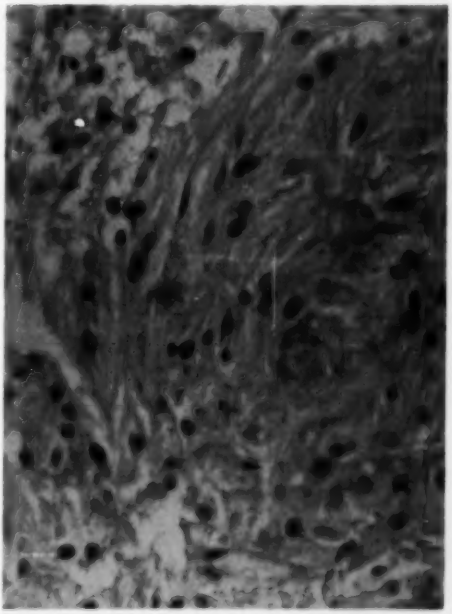

Fig. 4 (Morales and Hartz). Glial tissue partly filling the globe (×350).

## Discussion

It is obvious that the three layers of the small globe represent the three coats of the eye: the sclera, chorioid, and retina. The structure of the inner layer resembles the ciliary portion of the retina, but since further exploration was refused by the parents of the patient, it was impossible to determine if other parts of the retina were also represented. It was not possible to explain the presence of the glial cells and fibers inside the small globe; such cells and fibers are certainly not observed during the formation of the vitreous.

The vessels might be remnants of the vasa hyaloidea.

Since no lens and no cornea were found, it is tempting to try to explain at least part of the anomaly by the data provided by descriptive and especially experimental embryology.

It is well known that the ectoderm, which has the power to form the lens and possesses this power at a very early stage, may, even when it is transplanted to another site, form a small lens. This power receives a mighty impulse through induction by the optical vesicle. Early extirpation, before contact between this vesicle and the surface epithelium is established, prevents the formation of the lens in animals, with the exception of the rana esculenta.

The size of the lens is not only species bound but also depends on the size of the optical vesicle; experimental reduction of the size of the vesicle also causes a reduction of the size of the lens. It has also been demonstrated that the cornea is formed by the combined induction of the lens and the optical vesicle. It is interesting to observe that, if, after the formation of the cornea, the optical vesicle is extirpated, the cornea loses its differentiation and transforms into epidermis.[2]

The data enumerated may explain the absence of the lens and the cornea in our patient; they do not give a clue to the etiology of the malformation of the optic vesicle itself. We can only try to fix more or less exactly the period in which the developmental anomaly was produced: as the optical vesicles are formed during the third to fourth week of pregnancy and the separation of the lens from the surface and the differentiation of the retina begin during the sixth week, the anomaly was probably produced between these two points of time.[1, 4, 5]

It is not easy to classify exactly the malformation found in our case. Although anophthalmos is the complete absence of the eyeball from the orbit, clinically it is difficult to distinguish between anophthalmos and extreme microphthalmos. This can only be accomplished by the examination of serial sections through the contents of the orbit which is not always feasible. In our case different parts of the eyeball were found; therefore, anophthalmos did not exist.

When the development of the optical vesicle is arrested before its complete invagination, a so-called congenital cystic eye is formed. This is characterized by a sclera in which the tendons of the extraocular muscles are inserted in an abnormal way.

The cyst contains a yellowish albuminous fluid. The inner layer of the cyst is formed by a layer which resembles a misshapen retina.

There is also pigment epithelium and a thickening of this represents the anterior part of the corpus ciliaris. However, there is no pupil, no iris, anterior chamber, or vitreous body. Sometimes a small and degenerated lens is found in contact with the lower part of the cyst.[3]

In our case there was no cystic eye and the different layers of the small globe did not have the typical arrangement of this malformation; moreover, there were the two patches of skin—the most interesting finding in the present case. We therefore think that our case can best be classified as one of microphthalmos with transformation of the original "anlage" of the cornea into epidermis.

SUMMARY

In a seven-year-old boy patches of skin were found protruding between the eyelids. Exploration of one of the orbits revealed the presence of a small ocular globe. A biopsy was taken and microscopic examination showed the presence, in the wall of the small globe, of three layers which resembled the three layers of the normal eye. The small globe was partly filled with a mixture of glial and connective tissue. The case is considered to be one of microphthalmos with transformation of the anlage of the cornea into epidermis.

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# AN IMPROVED ELECTROLYSIS-IONTOPHORESIS UNIT*

S. I. ASKOVITZ, M.D.
*Philadelphia, Pennsylvania*

Within recent years, several papers have appeared describing devices for the electrolysis of distorted eyelashes and for effecting the iontophoresis of therapeutic agents across the cornea. The basic electrical requirement for either purpose is a source of direct current of about two ma. or less.[1]

Guyton[2] suggested having a length of wire attached to one terminal of a radio battery through a fixed resistance, with the second terminal to be grasped by the patient. No provision was considered necessary for regulating or measuring the current.

Selinger[3] devised a rather complicated instrument containing a number of different batteries, a voltage selector switch, a voltmeter as well as an ammeter, four terminal jacks, and so forth.

A relatively inexpensive power unit, originally assembled at this laboratory for certain experimental work, has been adapted so that it now serves ideally for either electrolysis or iontophoresis (fig. 1). The unit consists essentially of a 45-volt dry cell battery, a variable resistance (total 10,000 to 30,000 ohms), a small meter registering the amount of current (to five ma.), a pair of jacks for plugging in the wires, and an off-on switch.

The components are all contained in a small metal box which may be hung from a wall or used on a desk or table. The meter, in addition to indicating the strength of the current in use, provides a simple means for testing in advance whether the battery is in satisfactory condition (to avoid the embarrassment of an ineffectual current).

One wire is connected to a convenient hand electrode, consisting of an aluminum

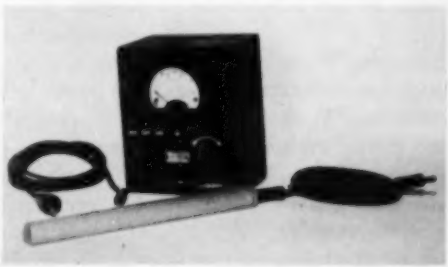

Fig. 1 (Askovitz). Electrolysis-iontophoresis unit.

The improved electrolysis-iontophoresis unit is being manufactured by the Locust Optical Company, 261 South 11th Street, Philadelphia, Pennsylvania.

* From the Ophthalmology Research Laboratory (Dr. I. H. Leopold, Director). Albert Einstein Medical Center, Northern Division. This work was made possible by a grant from the Weinstock Fund.

rod coated with a special corrosion-resistant material. The other wire is directly attached to an insulated spring clip, by means of which a very fine probe or other type of terminal may be securely grasped as the active electrode. For electrolysis, the wire to the patient's hand electrode is plugged into the positive pole, and the other wire to the negative pole. For iontopheresis, the position of the electrodes depends upon the chemical nature of the agent to be transferred.[4]

*Albert Einstein Medical Center (41)*

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## Ophthalmic Miniature

In the diagnosis of incipient cataract and incipient amaurosis, the catoptrical test is, in ordinary cases, decisive; for in amaurosis uncombined with glaucoma, the three images are distinct, while in even the early stage of cataract the inverted image is obscure or extinct. The diagnosis of incipient cataract and incipient glaucoma requires the catoptrical test to be familiar to the observer, else he may not distinguish that, when the candle is held in the axis of the eye, the inverted image is indistinct in both diseases; but whenever it is moved to one side, it becomes distinct in glaucoma; whereas in cataract, it either remains as obscure as before, or from the circumferential part of the lens being more affected than the central, it is obliterated.

Wm. Mackenzie, M.D.,
*Practical Treatise on the Diseases of the Eye,* 1854.

# SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

## CHICAGO OPHTHALMOLOGICAL SOCIETY

October 5, 1953

DR. GAIL R. SOPER, *President*

(The clinical meeting was presented by the Department of Ophthalmology, Northwestern University Medical School)

### CORECTOPIA

DR. WALTER E. HAGENS presented a 46-year-old white man, who stated that his vision had been poor all his life and that his eyes bothered him for many years. He had had many pairs of glasses but felt they did not help and had not been wearing them. Past history was noncontributory and there was no family history of a similar eye condition. He works in a metal products company.

Visual acuity was: R.E., counting fingers at three feet; L.E., 20/100. The left vision could be improved to 20/50 with +8.0D. sph. Corneal diameters were 11 mm. The pupils were markedly displaced upward and inward, were very small, and reacted only slightly to light. The iris was tremulous.

The slitlamp revealed vitreous in the pupillary opening. The right eye was somewhat injected and tension was 46 mm. Hg. (Schiøtz). Tension in the left eye was normal. The use of pilocarpine had no effect on the tension. A few days later the tension was normal, and has been on subsequent examinations although it probably fluctuates.

Ten-percent neosynephrine was instilled but the pupils dilated only slightly. The right disc could barely be seen and seemed normal. The lens was dislocated inferiorly and was cataractous.

According to Duke-Elder, the probable cause of this condition is the mechanical effect of abnormal and persistent vascular tissues associated with the irido-hyaloid vessels of the developing eye. The condition is bilateral, usually associated with other anomalies, and exhibits an hereditary tendency. Vision is poor and the eyes are frequently myopic. The iris is thin in the stretched segment and thick in the short segment. There is usually failure of development of the corresponding part of the ciliary body and zonule with ectopia lentis also.

*Discussion.* DR. EDWARD C. ALBERS said that these cases are difficult problems from the standpoint of therapy. In a patient recently seen, the pupils were dislocated down and nasalward and one could see vitreous in the pupillary area. The pupils could not be dilated and the margin of the dislocated cataractous lens could be seen across the middle of the pupil.

A broad iridectomy on both eyes was performed and in doing so with a pupil dislocated downward one removes most of the iris. The lenses were delivered intracapsularly without vitreous loss. The patient now has 20/30 vision in one eye and 20/50 in the other. He is quite sensitive to light but with a No. 2 tint in a cataract lens gets along well. Previously he had very poor vision.

### PITUITARY ADENOMA

DR. EMPOSTITO presented a case of pituitary adenoma.

*Discussion.* DR. DAVID SHOCH mentioned the classic work on pituitary adenomas done by Cushing about 30 years ago. The chromophobic adenomas of the pituitary involve the cells that have no real function, but the symptoms are the results of pressure on the chromophil cells of the pituitary, failing vision, bitemporal hemianopsia, optic atrophy, and the X-ray findings of an enlarged

sella; then there is the effect on the rest of the pituitary, the earliest and most common change being the change in sexual activity, impotency in males and, in females, cessation of menstruation.

The chromophobic adenoma must be differentiated from a group of three tumors that occur in this region. In addition to adenoma of the pituitary, there is the craniopharyngioma. The optic atrophy, the bitemporal hemianopsia, and the failing vision are the same and there is also enlargement of the sella turcica with these so-called Rathkepouch tumors. However, they occur in the younger age group, from 10 to 20 years, while chromophobic adenomas occur beyond the age of 30 years. Furthermore, the craniopharyngiomas very often form cysts, which grow out of the sella and, when they are slow growing, calcium is deposited in them and this calcification can be seen by X ray. This, plus the fact of the younger age, very often makes possible the diagnosis of craniopharyngioma.

The only other tumor in this area that must be differentiated is the suprasellar meningioma arising from the anterior lip of the sella. Although there are the same ocular signs there is no enlargement of the sella, and very often calcification is seen, depending on the length of time the tumor has been developing. There are no endocrine changes.

In this case there is one other point of interest; the elevation of intraocular pressure. There have been several reports of the association of elevated tension and adenomas of the pituitary, so that it is unlikely that it is simply a coincidental glaucoma. The mechanism of the elevated tension is unknown, but there are two possibilities: one, that the tumor grows enough to interfere with venous drainage of the orbit, producing congestion in the intrascleral plexus; or that the tumor extends to the hypothalamic region, interfering with the function of this area in controlling water and salt metabolism.

Dr. Gordon has raised objections to the first possibility because of the very adequate anastomotic venous drainage of the orbit and the second theory is the more likely one. Small changes in osmotic pressure in the blood can alter the intraocular pressure markedly and it may well be that involvement of the hypothalamus causes alteration in osmotic pressure.

### Chiasmal arachnoiditis

Dr. Hauser presented a case of chiasmal arachnoiditis.

*Discussion.* Dr. Daniel Snydacker: The question of chiasmal arachnoiditis has been discussed pro and con over a period of years. About 10 or 12 years ago it was described as a condition which resulted in poor vision with characteristically unusual field defects which could not be accounted for on any other basis. Many patients with this condition were operated upon, and nothing found except some thickening of the arachnoid in the area of the chiasm, which established the diagnosis.

Subsequently, as the condition was studied further, there was a question as to whether such a syndrome actually existed. It is fairly well agreed by ophthalmologists and neurosurgeons at this time that chiasmal arachnoiditis occurs rarely if at all.

In this particular case one wonders how removal of a piece of arachnoid 1 by 1 by 2 mm. could have had the effect of increasing the fields. Further, it would seem unlikely that if there were chronic inflammatory tissue of the arachnoid, merely surgery itself could eliminate the arachnoiditis. It would seem that the operative trauma might tend to increase the inflammatory reaction and further increase the size of the field defect. One might therefore cast some doubt on the validity of the diagnosis of chiasmal arachnoiditis.

Dr. Frank W. Newell: Heuer and Vail, in 1931, described opticochiasmic arachnoiditis as a definite entity. Arachnoiditis occurs in patients who are luetic and in others

who show some basis for an inflammatory reaction. But in general it is conceded that the isolated disease, occurring spontaneously and not complicating other infection, if it exists, is very uncommon. One might suggest, however, that this child had far better treatment for her visual difficulty than had the man with the proven tumor.

Recently a patient was seen who was blind in both eyes. Three years ago when seen by an ophthalmologist, he had had optic atrophy in one eye due to a chromophobe adenoma. He is now blind in both eyes with bilateral optic atrophy and there is no treatment of value. In the patient just presented, who was 56 years of age when diagnosis was first made and is now aged 60 years, removal of the tumor would preserve vision for the remainder of his life. If his physical condition is such that the tumor cannot be surgically removed, roentgen therapy to the pituitary gland might reduce its size so as to be compatible with continued vision. It is erroneous for ophthalmologists to observe these cases only, as they go gradually downhill until such time as therapy is not possible.

### Implantation cyst of iris

Dr. Milton M. Scheffler presented a case of traumatic epithelial implantation cyst of the iris in which the history of the perforating injury was quite obscure. The onset of symptoms and development of the cyst occurred many years later. Surgical interference resulted in an excellent recovery.

### Mechanism of primary glaucoma

Dr. H. Saul Sugar, Detroit, Michigan, (by invitation) presented a paper on "The mechanical versus the neurovascular schools in the mechanism of primary glaucoma," which is published in full on pages 645-652 of this issue of The Journal.

*Discussion.* Dr. Peter C. Kronfeld: Dr. Sugar has understated some of his own contributions to the subject under discussion, particularly the importance of the pre-existing narrowness of the entrance to the chamber angle in the glaucoma that we call the narrow-angle type. There has been recent evidence supporting this fact of pre-existence coming from an entirely neutral camp. New chamber depth measurements by the Rosengren school in Sweden indicate that the narrowness of the entrance to the angle is a genetic characteristic present in a significant percentage of the relatives with acute glaucoma.

Dr. Saul Sugar (closing): Dr. Hughes asked a question concerning the cyclitic crisis. This is a form of secondary glaucoma which I consider to be a special subheading under secondary glaucoma associated with uveitis. Little is known about the formation of aqueous in the secondary glaucomas, and possibly Grant's work will prove or disprove whether or not there is an increase in the secretion of aqueous. There is apparently no evidence as yet to disprove that secondary glaucoma associated with uveitis, or that associated with rupture of the lens capsule where there is lens material in the aqueous, or that type of glaucoma associated with posterior subluxation of the lens, are not associated with an increase in the production of aqueous.

The occurrence of acute glaucoma following the use of miotic drugs depends on the relationship between two factors: one which will cause an increase in resistance between aqueous outflow between the posterior chamber and the anterior chamber, and the other factor of pupil constriction. The effect will depend on which factor is greater. Most often the pupil-constricting factor is dominant, but there are instances of pilocarpine causing an acute attack of glaucoma in a patient who has had a previous attack which has been controlled.

Richard C. Gamble,
*Recording Secretary.*

## YALE UNIVERSITY CLINICAL CONFERENCES

### Ophthalmic manifestations in the neurologic patient

Dr. R. M. Fasanella, *presiding*

On October 14, 1953, Dr. David G. Cogan, Boston, director of the Howe Laboratory of Ophthalmology, Harvard University Medical School showed a series of lantern slides and moving pictures to illustrate the following ocular motor phenomena:

1. Lack of awareness of position of eyes in persons who are congenitally blind. This was interpreted as indicative of the absence of proprioception arising in the extraocular muscles.

2. Diagnostic sign of 4th nerve paralysis in the presence of a 3rd nerve paralysis. Intact 4th nerve function is thought to be present when there is intorsion of the affected eye on attempted downward gaze.

3. Internuclear ophthalmoplegia is due to lesions of the medial longitudinal fasciculus between the levels of the 3rd nerve and 6th nerve nuclei. The chief clinical signs are failure of adduction on attempted gaze to the side. The bilateral variety is most frequent with multiple sclerosis and the unilateral variety is most frequent with vascular lesions.

4. Unilateral cerebral lesions cause definite, although inconspicuous, ocular motor disturbances in the conscious patient. These include asymmetry in the opticokinetic response, saccadic following movements to one side, and lateral deviation of the eyes on forced closure of the lids.

*Discussion.* Dr. Fasanella: What is the operation of choice for ptosis in a Marcus Gunn phenomenon?

Dr. Cogan: As far as I know, any operation, as though there were no Marcus Gunn phenomenon.

Dr. Fasanella: At what level is a source of nystagmus in multiple sclerosis? Is it cerebellar?

Dr. Cogan: I think it is chiefly at the longitudinal fasciculus level.

Dr. Sam Black: Can we say that vertical nystagmus always denotes a superior colliculus level?

Dr. Cogan: No. Vertical nystagmus is most often due to a nonspecific lesion in the brain.

Dr. Black: What are some causes for monocular diplopia?

Dr. Cogan: (1) Partial sclerosis of the lens nucleus especially in myopes. (2) Displaced lens. (3) Psychogenic. I never saw one on a psychogenic basis.

Dr. Ernest Rosenthal: I saw one on a neurotic basis. This patient complained of seeing three headlights when driving at night and had marked difficulty in judging on which side of the lights to drive. She was seen in consultation and again no real cause could be found. She wore a mild minus lens for which coating of the lens did not help. This diplopia was present even without glasses. Ocular pressure was normal.

### Electroretinography

On November 13, 1953, Dr. J. Miles O'Brien, Bridgeport Connecticut, presented the subject of "Electroretinography."

*Discussion.* Dr. Fasanella: Should we all have an electroretinography apparatus in our offices?

Dr. O'Brien: No. Its primary use at present is in diagnostic and research centers.

Dr. Lovekin: Does the electroretinogram help in macular evaluation in cataract cases?

Dr. O'Brien: No. You may have a hole in the macula and a normal electroretinogram. It is helpful in detachment. It is a reflection of the total retinal activity.

Dr. Wong: What is the importance of the A-wave in prognosis?

Dr. O'Brien: Only in retinitis pigmentosa is the A-wave gone.

Dr. Wong: What is the effect of pressure on the eye?

DR. O'BRIEN: The B-wave becomes supernormal. This may also occur in glaucoma.

DR. YUDKIN: Has any work on the optic nerve been done?

DR. O'BRIEN: Dr. Granit has tested single nerve fibers in the optic nerve and retina.

DR. WONG: What does the electroretinogram show in amblyopia ex anopsia?

DR. O'BRIEN: Normal recording.

DR. LOVEKIN: Does that lead to the conclusion that the disturbance is cerebral, not retinal?

DR. O'BRIEN: Also that in amblyopia. It is chiefly the macula that is involved, which would not affect the electroretinogram. To study cone activity, one would have to work with flicker or pure chromatism, and even then rod waves may be superimposed.

DR. CLARKE: What creates the electrical activity?

DR. O'BRIEN: Light activity on the retina sets up the response. There are some resting currents in the eye without light, but no electroretinogram. With pressure on the eye, you can get a higher B-wave.

DR. BLAKE: That may be similar to the phosphene you get with pressure on the eye.

DR. GLASS: Do electromagnetic waves outside the visual spectrum produce an electroretinogram?

DR. O'BRIEN: I do not know of any work done along that line. It has all been done with light in the visual spectrum.

DR. FREEMAN: Is there any quantitative relation in retinal damage?

DR. O'BRIEN: Only in relation to the word "extensive." Small areas of retinal damage do not affect the electroretinogram.

DR. CLARKE: Does not cutting of the optic nerve affect the electroretinogram.

DR. O'BRIEN: There is a time factor involved. You would get a response for a time after cutting the nerve.

### DIAGNOSIS OF PRIMARY GLAUCOMA

On November 20, 1953, DR. WILLIS S. KNIGHTON, New York, discussed "aids in the diagnosis of primary adult glaucoma." An abstract of his paper follows:

Careful history is the most important first step with especial attention to acute attacks or insidious indefinite symptoms. Don't neglect the hereditary aspect.

Increased tension may be the first direct evidence of glaucoma, but is often missed at casual office visits, especially if they are always at the same time of day. The phase of the tension, whether ascending or descending, is best ascertained by a 24-hour study, but a compromise plan of seeing the patient during as much of the day as possible will often give valuable information.

Attention is called to the factors which influence the interpretation of the intraocular pressure (for which the tension reading is only an indication) such as corneal curvature, ocular rigidity, reaction of the patient, and so forth.

Even when the eye appears to be normal, a study of the coefficient of aqueous outflow may indicate glaucoma long before there is any other clinical evidence. This coefficient is always reduced in untreated open-angle glaucoma but is always normal in narrow-angle glaucoma between attacks. It can be improved by medication or surgery. An electronic tonometer is necessary for this study which is now considered essential.

Of the numerous provocative tests only two are in general use: the darkroom test for suspected narrow-angle glaucoma and the water-drinking test for suspected open-angle glaucoma. Dilatation with mydriatics may precipitate an attack in the former and is useless in the latter. The water-drinking test is negative in narrow-angle glaucoma but gives a positive increase in tension in open-angle glaucoma in 75 to 90 percent of the cases.

Gonioscopy is essential to a thorough understanding of each case. The angle may appear open in narrow-angle types between attacks but is always closed during an attack. Suspicious narrowness between attacks may prevent indifferent treatment even though an

absolute diagnosis cannot always be made. After known acute attacks, peripheral anterior synechias should be suspected and looked for, knowing how they modify the future course and its treatment.

Unfortunately the angle may also appear narrow in open-angle glaucoma in which it never closes. Peripheral anterior synechias should not appear in these eyes. The diagnosis must be made on the basis of other findings. Generally the angle appears wide open and clean.

Field changes do not appear early in either type of glaucoma. The first demonstrable defect is supposed to be a vague scotoma between the upper pole of the blindspot and the fixation point. Later it is attached to a wing extending from the blindspot but even in early stages it is claimed that a connection can be demonstrated by using a blue test object. Special techniques may eventually prove to be more revealing than the usual procedures but, at present, nothing replaces careful painstaking studies.

Field defects develop more slowly in narrow-angle glaucoma unless there are very frequent episodes, but they are common in open-angle glaucoma where a sustained, although moderate, elevation has persisted for some time.

Early glaucoma cannot be diagnosed by field defects alone. It must also be realized that opacities in the media, for example, cataract, can modify the field obtained. Miosis due to drugs and uncorrected refractive errors must also be considered.

Glaucomatous cupping of the optic discs must be differentiated from a physiologic cupping, atrophic cupping, coloboma, cavernous atrophy, and congenital pits of the nervehead.

Narrow-angle glaucoma tends to show pallor or atrophy of the nervehead before deep cupping appears; whereas, the reverse is found in open-angle glaucoma.

Glaucomatous cupping is a late manifestation.

As aids in diagnosis the above tests or examinations are presented in the order in which they may give evidence of early glaucoma. Each one makes its own important contribution to the whole picture and none should be omitted in a proper study of glaucoma.

*Discussion.* DR. EUGENE BLAKE: There are several points I would comment on. I would like to ask Dr. Knighton whether he finds the combination of sympathomimetic and parasympathomimetic drugs useful in treatment of glaucoma. I believe we should take the tension of everyone over 40 years of age, but we must be careful of the interpretation of the initial tension in a nervous patient. I feel the aqueous-flow test is of great value in the study of glaucoma. I agree with Dr. Knighton that the water-drinking test and the darkroom test are the most useful of the provocative tests. And in regard to gonioscopy, I use the head of the slitlamp for magnification.

DR. KNIGHTON: Most miotics are vasodilators, and I believe some acute attacks of glaucoma may have been precipitated by pilocarpine. I occasionally prescribe adrenalin drops, followed in a few minutes by pilocarpine, or I may prescribe one or two-percent pilocarpine made up in adrenalin (1:1,000).

DR. YUDKIN: I have gotten into difficulties with the use of adrenalin, and no longer use it. I believe the aqueous-outflow studies will prove very valuable.

DR. KNIGHTON: Adrenalin can be used as a provocative test, as done by Knapp. I use adrenalin occasionally and think it is safe if a miotic is used two or three minutes later. I never prescribe eserine for use at home because of its irritating properties. It used to be said that if the patient were not controlled by pilocarpine alone, operation was indicated. Now it is possible in individual cases to control the tension with adrenalin and pilocarpine, or sometimes with furmethide.

DR. L. LOVEKIN: Is there any response to the use of drugs on the outflow test?

DR. DE SUTO-NAGY: I want to re-emphasize the value of studying the diurnal variations in tension. Does Dr. Knighton feel it is replaced by the study of aqueous flow?

DR. KNIGHTON: Phasing studies are not replaced by aqueous-flow tests. Usually in chronic simple glaucoma the aqueous flow is constant all day long, but it is affected by medication. Therefore, phasing tests may help to regulate the use and timing of drug dosage.

DR. W. GLASS: Dr. Knighton mentioned the studies in which several cases continued to lose field after the tension had been reduced to normal levels around 22 mm. Hg (Schiøtz) with miotics. In these patients the aqueous outflow studies were still abnormal, and it was found that increased miotic dosage resulted in improving the outflow figures and halting the loss of field. Assuming the increased miotic dosage in a similar case failed to improve the outflow figures, would Dr. Knighton feel this was sufficient indication for operation, even with tension of 22 mm. Hg?

DR. KNIGHTON: That brings up the whole question of the so-called low tension glaucoma and its therapy. I do not know the pathogenesis of glaucoma, and can only recommend that you read Dr. Becker's paper in a recent issue of THE JOURNAL to learn the present state of our knowledge of outflow studies in regard to the levels of intraocular pressure and control of glaucoma.

Dr. William I. Glass,
*Recording Secretary.*

## OPHTHALMIC MINIATURE

The subject of this case was an old man named Rumbold, the father of a well-remembered beadle at the London Hospital College thirty years ago. . . . He was upwards of eighty, and almost in his dotage. I was asked to see him because, as I was told, he had red "streaks on his head" which were painful and prevented his wearing his hat. . . . The "red streaks" proved, on examination, to be his temporal arteries, which on both sides were found to be inflamed and swollen. The streaks extended from the temporal region almost to the middle of the scalp, and several branches of each artery could be distinctly traced.

Jonathan Hutchinson,
*Archives of Surgery*, 1:325, 1890.

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## THE XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

MONTREAL—NEW YORK
1954

After a lapse of 80 years, the North American continent was again host to the International Council and Federation of Ophthalmology with a numbered congress. It has been a most successful venture, particularly from the scientific side, and an experience that will be long remembered by the many from throughout the world who attended it. The success is due to the hard work and the great executive ability of William L. Benedict, the secretary-general, and his devoted, loyal, and efficient Canadian and American committees, whose members gave of themselves unstintingly to the tasks to

which they were assigned over the several years of planning that were required to put the affair over.

Our Canadian colleagues deserve special credit for their part in the congress. This was a part that was suddenly thrust upon them on short notice, creating difficulties in rapid organization, which all of those who attended the Montreal session realize were extraordinarily well and happily solved by the Canadian organizing committee under the efficient direction of G. Stuart Ramsey, the associate secretary, and with the assistance of Mark Marshall, the president of the Canadian Ophthalmological Society.

The congress officially opened in Montreal on Friday morning, September 10th, in the beautiful auditorium of the University of Montreal. This new and handsome edifice with spacious halls and marble corridors gave a background of dignity and prestige to the pageantry and color of the opening ceremony. The flag-draped stage, in the back of which hung the official blue and gold flag of the congress, was the heart of the setting. After the audience was seated, the official delegates representing countries, organizations, universities, and ophthalmic societies from over 50 countries of the world, marched in, capped and gowned in their multicolored academic robes, many wearing their decorations, and took their seats in the front of the hall facing the stage. Then the platform group consisting of the officers and members of the International Council, the officers of the congress, headed by Bernard Samuels of New York, the president, and other dignitaries filed onto the platform and took their places facing the audience. The color of the hoods, robes, and flags, the beauty of the hall, the importance of the occasion, and the solemnity of the moment suddenly seemed to create an aura of international friendship and goodwill that persisted throughout the entire congress.

After the invocation by Monseigneur Maurault, Rector of the University of Montreal, Dr. Samuels introduced Mr. Paul Martin, the Minister of Health of Canada, who gave an inspiring talk on the importance of ophthalmology to the health and happiness of people and described some of the steps that the government of Canada is taking to improve and to foster ophthalmic education, training, and care. Sir Stewart Duke-Elder then replied for the council, following which Mr. Martin conferred the Gonin Medal, sponsored by the University of Lausanne, Switzerland, upon Sir Stewart, amid prolonged applause. The choice of this year's recipient was obviously a popular one and most suitable. Had a poll been taken of every ophthalmologist in the world one can wager that Sir Stewart would have been the unanimous choice for this great honor which crowns the recipient with the unwritten title of "World Ophthalmologist Number One." Then followed short addresses of welcome from Monseigneur Maurault; Dr. Lyman Duff, dean of medicine of McGill University; Dr. Mark Marshall, president of the Canadian Ophthalmological Society; and of selected delegates: Dr. Weve of Holland, representing Europe; Dr. Escapini of El Salvador, representing North America; Dr. Valdeavellano of Peru, representing South America; Dr. Mahmond Adel-Haniel Attiah of Egypt, representing Africa; Dr. Nakamura of Japan, representing Asia; and Dr. J. Ringland Anderson of Australia, representing Australasia.

Dr. Bernard Samuels then gave his presidential address on the subject of "Medical training in the United States." The ceremony closed with the official address by the president of the International Council, Sir Stewart Duke-Elder, following which the delegates, members, officers, and guests filed out of the building for the taking of the official photograph of the congress on the steps of the central building of the University of Montreal.

In the afternoon the scientific session, held in the Physical Sciences Centre, McGill Uni-

versity, began with a panel discussion on "Retrolental fibroplasia" for the entire membership. After this, two sessions were held simultaneously with an excellent program of approximately seven papers arranged for each. At the same time the International Organization against Trachoma, under the presiding of A. F. MacCallan, held its scientific session with seven presentations. In the evening, a reception and collation for the members and guests of the congress was given by His Worship The Mayor Camillien Houde, in the lovely Chalet du Mont-Royal overlooking the beautiful city of Montreal. It formed an unforgettable picture of great charm.

On Saturday, September 11th, the sessions began with a joint panel discussion on "Virus diseases of the eye." Following this the sessions were again divided into two, simultaneously meeting, each with an excellent program. Luncheon was served in the spacious great hall of McGill University and the members and guests were then free to go sight-seeing or proceed to New York for the next part of the congress. In view of the alarming rumors of the Hurricane Edna, there was considerable speculation as to whether New York would still be standing by the time the congress reconvened. Fortunately, for New York and the congress, these rumors were without fact.

### New York
### September 12th-17th

On Sunday afternoon, September 12th, a reception for the members and guests was held in the headquarters of the congress, the extraordinary Waldorf Hotel in New York, following which an illustrated lecture "A journey to the Never-Never" was given by Miss Ida Mann, now residing in Australia. A large and highly entertained audience listened to her describe her experiences and adventures, ophthalmic and otherwise, in the wilds of Australia.

The congress officially reconvened on Monday, September 13th, with an assembly in the Grand Ballroom of the Waldorf. After the call to order by the president of the congress, an address of welcome was given by a deputy for the mayor of New York, Robert F. Wagner, who was unable to be present. Dr. Alexander T. Martin, president of the New York Academy of Medicine, graciously extended official greetings from the medical profession of New York. He was followed by our own Dr. Alan C. Woods who gave a most moving and eloquent welcome on behalf of American ophthalmology. Dr. Detlev Bronk, president, the Rockefeller Institute for Medical Research, then gave a noble address, the theme of which was the need for freedom of science and scientific methods and the universality of science. Make a note to be sure to read it carefully when it appears in the *Acta* of the congress. Sir Stewart Duke-Elder then gave an official response as president of the International Council.

The presidential luncheon, sponsored by the officers and committees of the congress, was well attended and enjoyed by a large number of official delegates and members of the various working committees.

Following the luncheon, the congress got down to official scientific business, and for the duration of the event, one was kept busy every moment trying to take in the multi-ringed circus. Each day there were three simultaneous scientific sessions each with a program of eight to nine papers, a total of about 200. Each day, also there were televised (in color) surgical operations, the crowded attendance at which testified to their great popularity. Also each day cinema demonstrations of surgical procedures and other items of ophthalmic interest were given in a crowded hall. Each hospital in New York daily put on surgical clinics that were widely attended and appreciated. The International Association for Prevention of Blindness held its scientific meeting on Monday, September 13th. Symposia in experimental ophthalmol-

ogy were held, one on the "Transparent media of the eye" and the other on "Tonography and aqueous-humor dynamics," each attracting large audiences. The official subjects of the congress (a) "Primary glaucoma" and (b) "The etiology of uveitis" were discussed in the Grand Ballroom which was crowded to overfilling. Official and unofficial discussions of these papers continued to the last minute, with many speakers seeking an opportunity to express their views still waiting their turn.

Indeed, the enthusiasm of the members, their almost insatiable appetite for scientific knowledge, and their energy in getting about from one meeting room to the next was one of the extraordinary phenomena of an extraordinary congress. Every event seemed to be crowded with members and guests. The aisles of the scientific exhibits, all of which were remarkably good, and of the technical exhibits were always, it seemed, in need of some sort of traffic-control lighting.

The social side of the congress was also most noteworthy. There were two official subscription dinners, the one on September 13th was presided over by Dr. Everett L. Goar and the speakers were A. B. Reese, Barrenechea, Bietti, Paufique, Moffat, followed by the guest speaker, Helen Keller, who was magnificent. The other dinner was on September 15th and was presided over by Prof. H. J. M. Weve of Holland. The speakers were Dunphy, Arruga, Valdeavellano, Ehlers, and the guest speaker Morris Frank of the Seeing-Eye Foundation. Each of these dinners was crowded to capacity and happily enjoyed.

The official banquet was held on September 16th and was a truly lovely party. The president of the congress gave a short address of welcome followed by Sir Robert Scott, Minister of the British Embassy in Washington, who gave a most entertaining and witty "Toast to ophthalmology." He compared ophthalmology with diplomacy sometimes to their mutual advantage, sometimes not, with a sparkling play of fantasy. The responses to the toast were given by Dr. Marcel Kalt of Paris for the French-speaking members of the congress and by Dr. Moacyr Alvaro for the Spanish-speaking members. Their addresses were beautifully presented and thoroughly appreciated. Then followed dancing to a splendid band, and lovely ladies from every part of the world added their beauty to an unforgettable evening.

Besides these official social functions our New York hosts overwhelmed their friends and acquaintances with gracious formal and informal dinners, theater parties, cocktail parties, luncheons and so on, so graciously that it is believed that every foreign member of the congress had the opportunity of meeting socially with his American colleagues.

The closing ceremony of the congress took place on the afternoon of September 17th. The delegates and officers clothed in their academic robes and hoods, as before, paraded into the ballroom and listened to farewell messages from the president of the congress, the president of the council, and chosen delegates from various hemispheres. These were Arruga of Spain, representing Europe; Puig-Solanes of Mexico, representing North America; J. Verdaquer Planas of Chile, representing South America; H. D. Dastoor of India, representing Asia; Hadi Rais of Tunisia, representing Africa; and J. Ringland Anderson of Australia, representing Australasia.

Sir Stewart reported on the activities of the council and the federation and described the new edition of the *Index Ophthalmologicus* that had just come off the press. This is a fine book containing the lists of ophthalmologists by countries throughout the world. It is pleasing to find that the list of those in the United States is quite complete. It should be a useful directory for many people. He also mentioned that a dictionary of ophthalmic terms, in English, French, German, Spanish, Italian, and Latin, is in the

process of completion, under the direction of the International Council, and should soon be available for use. It is good to know that Duke-Elder remains the president of the International Council for another four years. Before closing the congress, Sir Stewart paid glowing tribute to the great work of William L. Benedict in seeing the congress through to such a successful conclusion. And so ended another historic congress that will be long remembered for its many sided, eminently satisfactory activities.

The next congress will be held in Brussels, September 7 to 12, 1958. This will represent the centennial celebration of the congress in the place where it was founded.

It is difficult to find out exactly how many registered for and attended the XVII Congress but it is estimated that approximately 3,000 members and associate members actually attended all or a part of the congress. There were official delegates from 52 countries including the U.S.S.R. and other "iron curtain" countries, and it is a good thing when we can all come together in harmony and peace and exchange scientific ideas of such great benefit to people everywhere.

While the final curtain has been rung down, there still remains a lot of clean-up work for the committees to do. The chief jobs yet remaining are (a) that of the finance committee and (b) that of the editorial committee. The editorial committee under the able chairmanship of Francis H. Adler, has a monumental task ahead to prepare and to publish the official transactions or *Acta*. Each author of a paper is urged to co-operate fully with Dr. Adler and members of his committee in order to lighten their burden and to speed publication. The committee on finance is faced with the responsibility of finding ways and means to finance the printing, publishing, and distribution of the *Acta*, a very expensive business, but one that is of the utmost importance and one that must be done properly. It will be at least 80 years before another International Congress of Ophthalmology comes our way again. This particular congress is a burden of our time that must be cheerfully assumed by all of us. In the words of Kipling's Absent Minded Beggar—"Pass the hat for your credit's sake and pay—pay—pay." More about this later.

Derrick Vail.

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## THE 1954 ACADEMY MEETING

The 59th annual session of the American Academy of Ophthalmology and Otolaryngology was held in New York, September 19th to 24th, following the meeting of the International Congress. Well known for the excellence of its scientific program, it did not suffer by comparison with the International Congress, there being the usual number of outstanding papers. There was a marked contrast in the attendance of the sessions of the two meetings. As appears to be the custom, at the meeting of the International Congress, the actual attendance at some of the major meetings was very small, while all of the sessions in the Grand Ballroom were well attended at the academy meeting.

In his presidential address, Dr. Walter Theobald stressed the important part that the newly developing medical centers are playing in medical education. The guest of honor was Dr. William James McNally; his address on "Some thoughts about research in the training of a doctor" should be read repeatedly by all teachers and all physicians who present papers at medical meetings. The symposium, "Antibiotics up-to-date," was presented by Dr. Irving H. Leopold, Dr. James Maxwell, Dr. Seymour Halbert and Dr. Wesley W. Spink. It was interesting to learn that this group feels that pencillin is still the antibiotic of choice; it was stated that less than two percent of patients are subject to toxic effects from it.

The Jackson Lecture, sponsored by the Ophthalmic Publishing Company, was presented by Dr. Algernon B. Reese of New York; his subject was "Persistent hyperplastic primary vitreous." The lecture clearly differentiated this condition from retrolental fibroplasia with which it has been confused in the past.

Among the papers presented, were: "The treatment of toxoplasmic uveitis," by Dr. Ralph W. Ryan, Dr. William M. Hart, Dr. John J. Culligan, Dr. Ralph Gunkel, Dr. Leon Jacobs, and Maryjane K. Cook; "Subacute circumscribed macular choroiditis simulating intraocular tumor," by Dr. Wilfred E. Frey and Dr. Edmund B. Spaeth; "New lens material: Indications and hazards," by Dr. Arthur Kenney and H. Lyle Duerson, Jr.; "The effect of retrobulbar injection of procaine on the optic nerve," by Dr. Frank D. Carroll and Dr. Andrew deRoetth, Jr.; "Familial primary hyperplasia of the orbital margin," by Dr. Alberto Urrets-Zavalia, Jr., of Argentina; and "The multiple pattern method of visual field examination," by Dr. David O. Harrington.

On Friday morning, a symposium was held on "Ocular allergy" in co-operation with the American Society of Ophthalmic and Otolaryngologic Allergy under the direction of Dr. A. D. Ruedemann, Sr. Others taking part included Dr. George L. Waldbott, Dr. S. Albert Hanser, Dr. Frederick H. Theodore, and Dr. Erling W. Hansen. Dr. Forest J. Pinkerton presented a paper making "A plea for the best possible preinduction physical examination."

Two symposia deserve special mention. During the symposium on "Retrolental fibroplasia" led by Dr. Algernon B. Reese, Dr. William C. Owens, Dr. Jonas S. Friedenwald, Dr. William A. Silverman, Dr. V. Everett Kinsey, Dr. Arnall Patz, and Dr. Frederick C. Blodi, the clinical course, pathology, pediatric considerations, etiology, animal experimentation, and management of the disease were discussed. It was a striking example of how, by co-operation with each other, the clinician (both general and ophthalmic), the pathologist, and laboratory research workers can solve some of our problems. The presentation seemed to prove beyond doubt that, on the basis of the work done by this group and others, excessive oxygen given to premature babies plays a major role in the production of the disease.

The second symposium, on "Congenital glaucoma," was presented by Dr. Robert Shaffer, Dr. Harold Scheie, Dr. Otto Barkan, Dr. Joseph S. Haas, and Dr. Samuel J. Meyer. The symposium discussed the pathogenesis, clinical courses, goniotomy, other types of surgery, and the results in congenital glaucoma. This symposium, another example of how it is possible for authorities with divergent views to come together and reach an agreement on certain basic factors, will help toward better understanding of the prognosis and proper care of these cases. These academy sponsored and financed symposia are rendering a great service to American ophthalmology.

Clinicopathologic case reports were presented by Dr. S. Rodman Irvine and Dr. Warren A. Wilson, Dr. Arthur E. Schultz, Dr. Theodore E. Sanders, Dr. W. G. Klingberg and Dr. H. G. Slaughter, and Dr. J. Jackson Stokes. These short, interesting case reports add a great deal to the meeting and seem to help bring the members to the meetings on time.

The usual interesting motion pictures were all on surgical subjects and included: "The spoon extraction of cataract," by Dr. Norman L. Cutler; "A simplified ptosis operation," by Dr. Charles E. Iliff; "Resection of levator via external route," by Dr. Brendan D. Leahey; "Goniotomy," by Dr. Otto Barkan; and "Reconstruction of new lower lid following entire removal for carcinoma of lower lid," by Dr. Wendell L. Hughes.

There were many outstanding scientific exhibits. Lack of space necessitates that mention be limited to those receiving awards.

The first award was given to Dr. David D. Donaldson for his exhibit, "The angle of the anterior chamber and related structures: Studies by means of stereophotography." It presented stereoscopic kodachrome pictures of the anterior chamber photographed by an ingenious stereoscopic camera designed and built by the exhibitor. The second award went to Dr. C. C. Teng, Dr. R. Townley Paton, and Dr. Herbert M. Katzin for their exhibit "Connective-tissue degeneration in the chamber angle." The work was done on Eyebank eyes and may offer the explanation for the increased resistance to outflow of aqueous in wide-angle glaucoma. The third award was given to Col. Victor A. Byrnes, Capt. D. V. L. Brown, H. W. Rose, and Paul A. Cibis for their exhibit on "Chorioretinal lesions due to thermal radiation from atomic bombs." Their work brought out a hitherto unsuspected ocular complication of atomic warfare.

Instruction courses have become an important part of the academy's program, and this year it was up to the usual standard. There were 54 continuous courses and 104 individual courses in ophthalmology alone. There were a number of new, younger men giving their first courses, so that it is apparent that the secretary of instruction in ophthalmology is developing teachers for the future. As usual, the courses were well attended. However, a newcomer in the field of instruction must be given serious consideration by the officers in charge of instruction in the academy. For the first time in the history of the academy, surgical procedures were televised at the time of the instruction courses. There is no question of the fact that these spectacular demonstrations do detract from the instruction courses, and it will be necessary to evaluate the importance of these demonstrations in relation to the value of the courses given.

There were 5,858 fellows, guests, residents, candidates, technicians, and ladies registered. The next annual meeting of the academy will be held at the Palmer House, Chicago, October 9-14, 1955.

Frederick C. Cordes.

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## CANADIAN OPHTHALMOLOGICAL SOCIETY

### 1954 Meeting

The annual meeting of the Canadian Ophthalmological Society was held in Montreal in the Physical Sciences Building of McGill University on Thursday, September 9th, immediately preceding the XVII International Congress of Ophthalmology. For this reason the meeting was attended by an unusually large number of distinguished guests. Two invited guests presented papers.

Dr. John H. Dunnington, of New York, spoke on "Defective ocular wound healing." This was an extension of his previous experimental and clinical studies, in which he has shown that minor imperfections in healing may lead to major complications following cataract extraction. His present studies show that hyphema and iris prolapse are largely due to defective healing of the wound, which in turn may arise from faulty closure of the wound and inaccurate placement of the sutures.

Dr. G. P. Sourdille of Nantes, France, presented an excellent discussion of "Lamellar keratoplasty," pointing out not only its value in improving vision but also as a therapeutic measure. He prefers a plastic contact glass to coaptation sutures for holding the graft in place. Professor Franceschetti of Geneva discussed this paper at some length, emphasizing the therapeutic value of such grafts. He showed excellent slides illustrating this aspect.

Of the members of the society, Dr. Rene Charbonneau of Montreal presented a clinical study of "Heredodegeneration of the macula (Stargardt's disease)," in four mem-

bers of the same family—a mother, two daughters, and a son.

Dr. J. S. Crawford of Toronto gave a report on "Repair of ptosis by using the frontalis muscle and fascia lata." He reviewed the results obtained in 73 children. In 59 this operation was carried out, while in the remainder levator resection was used in all except one where a tarsal resection was carried out. He felt the fascia-lata sling gave the best results. The operative technique was illustrated with a moving picture in color. Dr. R. M. Ramsay of Winnipeg reported on "Cataract extractions with the use of curare." He discussed a series of such cases corroborating Kirby's findings that curare akinesia greatly facilitates the operation and reduces operative complications and, in careful hands, is a safe procedure.

Dr. J. V. V. Nicholls, Dr. W. Turnbull, and Dr. K. A. Evelyn of Montreal reported on one phase of their studies of vascular hypertension. In their paper, "The caliber of retinal arterioles in normal and hypertensive patients," they estimated the relative A/V ratio in 200 normotensive and 100 hypertensive patients, grading the A/V ratio in comparable vessels in each quadrant in a 1-to-10 gradation system. In some cases the patients were examined by one observer while in others one observer checked against the other. A relatively high degree of agreement was obtained. The data agreed remarkedly well with those of Bjork, which were determined by a different method. It was found, as would be expected, that the A/V ratio was lower in hypertensives than in normotensives, but the difference was disappointingly small relative to the error of measurement. In general the A/V ratio correlated poorly with various indices of the severity of the hypertensive disease.

Dr. A. Lloyd Morgan and Dr. Maria J. Arstikitis of Toronto made a preliminary report of their extended study on "Concomitant strabismus in children." They found it difficult to determine the efficacy of surgical treatment. Patients who were thought to be cured in the first postoperative years sometimes became overcorrected later, and undercorrected patients later became straight. The percentage of cures, that is fusion without glasses, was very low.

Dr. Marc Plamondon and Dr. C. Auger of Quebec presented an interesting case report of a neuronevus of the choroid.

Dr. Clement McCulloch of Toronto presented a report of his experimental and clinical studies on the "Application of positive pressure to the orbit." These studies were carried out by placing a plastic bag under an ocular bandage and attaching it to a manometer. It was found that such a bandage loosens quite rapidly for about one-half hour and then quite slowly but constantly over the next one or more days. By means of the bag, pressure was applied to the orbit in other cases. It was found that a pressure of 65 mm. Hg may exsanguinate the lids and cause sloughing, while 25 mm. Hg prevents postoperative edema, but 10 mm. Hg has little or no effect. This disarmingly simple procedure would appear to have wide applicability.

Dr. R. G. Murray of Chapel Hill, North Carolina, spoke on "Ocular nerve palsies in children." He presented a statistical analysis of a large series of patients and stressed the great difference in the etiology of the condition in adults as compared to children. In the latter, inflammatory causes are in the minority and neoplastic in the majority. Dr. Murray pointed out the importance of careful and accurate study of all cases of strabismus in children, sudden changes in the angle requiring particular care.

John V. V. Nicholls.

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## PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY

### III Interim Meeting

The III Interim Congress of the Pan-American Association of Ophthalmology was

held in São Paulo, Brazil, June 11 to 14, 1954.

A unique feature of this congress was the system of simultaneous translation with qualified translators which made it possible for those who spoke different languages to understand the papers. The translation equipment consisted of a small radio receiving set and a little case with a battery that is hung around the neck, the shoulder strap serving as an antenna. In this way all papers can be listened to outside the meeting room, as all one has to do is to put on the earphones and tune in to the language that is best understood. Many were the colleagues whom we met in São Paulo in the corridors of the Medical Association building listening to the papers because they could not get into the crowded room. For the first time English-speaking attendance was excellent during the presentation of Latin-American papers and vice versa.

From the scientific viewpoint this congress was of great value, good papers being given by world famous colleagues. The following were of special interest:

The symposium on "Uveitis," in which the etiopathogeny, clinical diagnosis, clinical evolution, complications, and treatment were discussed. Perhaps there are few subjects in modern ophthalmology that are so in need of study for the most diverse opinions still prevail. However, it now seems certain that chronic endogenous uveitis (the most common type) can, from the etiologic point of view, be divided into two well-defined groups: (1) Granulomatous uveitis in which the causative agent lies within the eyeball, thus directly causing the lesion; and (2) nongranulomatous uveitis in which the causative agent or its toxin sensitizes the uvea, an allergic reaction being caused later by the entrance of the toxin into the uvea through the blood stream. This is a very important fact because, in the case of granulomatous uveitis, one should seek to exterminate the causative agent by specific or nonspecific means, whilst in the nongranulomatous cases one seeks to eliminate the causative focus. Antibiotics are of no use in the nongranulomatous forms. They can even aggravate the ocular condition by liberating a larger quantity of toxin which aggravates the allergic reactions or causes new ones. Desensitization with specific vaccines for the 40-odd strains of streptococcus pathogenic for humans is the treatment of choice. An antiallergic medication such as ACTH or cortisone is also indicated.

The subject of corneal transplants was also opportune, as this phase of surgery fascinates the ophthalmologist and the public. It was certainly useful for Latin American ophthalmologists to be able to compare notes on their results and take the opportunity to confer with colleagues from other countries. The history of transplantation of the cornea, biology of the graft, perforating grafts, and lamellar grafts were specially dealt with. This subject, that was an official theme of the Brazilian Congress of Ophthalmology, was also discussed in free papers at the Pan-American Congress.

One of the most interesting discussions was on recent advances in ocular therapeutics. This subject was subdivided into several sections. Thus, in the section on diseases of the orbit it was emphasized that the results of the treatment of exophthalmic goiter with radioactive isotopes are comparable to those obtained with extirpation of the thyroid.

Regarding allergic diseases of the cornea and sclera the favorable action of cortisone was emphasized. Mention was also made of the use of cauterizations with ether for corneal herpes. Parenchymatous keratitis is also thought to be allergic in nature and subconjunctival injections of cortisone give excellent results.

Lamellar grafts seem to give better results than perforating grafts (65 percent for the former; 40 percent for the latter). The smaller the diameter of the graft the better the results. The treatment of astigmatism by

Sato's method of retrocorneal incisions does not seem to give consistent results. In the section dealing with ocular tumors, it was emphasized that progress is being made in the field of differential diagnosis between a simple retinal detachment and a detachment caused by intraocular tumor, which is sometimes difficult. The technique consists of injecting isotopes into the blood and, by means of a Geiger counter that reaches the site of the suspected tumor, verifying whether it is a tumor or a simple retinal detachment.

The treatment of retinoblastomas by irradiation was also emphasized. Forty percent of all cases of retinoblastoma are bilateral. The nondifferentiated cells of retinoblastoma are sensitive to gamma rays in spite of their origin in tissue that is nonsensitive to irradiation. This explains the success of Reese's and Stallard's methods, the first author using roentgen rays, and the second small capsules of radium. It was also emphasized that an early diagnosis is important.

Regarding acute inflammatory glaucoma or narrow-angle glaucoma, the greatest progress in therapy lies in the appearance of adrenergic-blocking drugs that cause miosis by relaxing the dilator muscles of the pupil. Dibenamine, the most frequently used of these drugs up to a short time ago, has now given place in the experimental stage to Dibenzyline and to Diamox.

Peripheral iridectomies have given good results in recent cases. Malignant glaucoma, in which the anterior chamber does not reform after the operation, demands immediate extraction of the lens even if it is transparent. Several peripheral iridectomies prevent closure of the iridocorneal angle.

In infantile glaucoma surgery should be performed as early as possible. Iridencleisis or, preferably, Barkan's gonitomy, are the operations of choice.

The medical treatment of cataract was included in the program in order that it might be made clear that it was useless, in order to counteract theories that had been widely diffused and that are prejudicial to the unwary.

Syphilitic optic neuritis should be treated early with doses of penicillin of six million units. When visual acuity is lower than 20/50, treatment always seems to be useless.

In cases of optic neuritis due to methyl alchohol the lesion is caused by acidosis (excess of $CO_2$—more than 25 millequivalents per liter). Therefore, treatment should consist of administration of bicarbonate of soda intravenously and then by mouth.

Progress in the optical correction of ametropias and amblyopias was also considered, the most noteworthy contribution being modern contact lenses and the combination of minus contact lenses with plus lenses in the spectacles, thus creating a field glass on Gallileo's principle. The use of plastic lenses after cataract operations cannot, as yet, be recommended.

Medical treatment of squint was discussed and the value of orthoptic treatment in cases in which it is indicated was emphasized. The surgical treatment of strabismus was also reviewed; it was stressed that surgery is only adjuvant to medical treatment.

Ophthalmologists are returning more and more to general anesthesia for their operations. Thus, the description of the new techniques of anesthesia, with potentializing substances, antihistaminics, similar to 3277RP or not, such as 4560RP, aroused great interest. These drugs, besides increasing the action of analgesics and hypnotics, cause a reduction of metabolism, decrease of capillary permeability and amnesia; 4560RP causes the so-called pharmacodynamic leukotomia, the patient being completely indifferent to his environment. Artificial hibernation was also discussed. This consists in the blockage of the nervous system at all its levels and in the reduction of basal metabolism, this being completed by the application of ice over a large area of the body. In ocular surgery, potentialization of anesthesia has the following advantages: lowering of

the ocular tension which parallels lowering the blood pressure, decrease of ocular bleeding, easy ocular motility, and absence of postoperative vomiting.

Regarding retinal detachment, it was found that there are three essential conditions: (1) Discovery and localization of the tears, which can be best obtained with a binocular ophthalomoscope for indirect ophthalmoscopy combined with a hook to compress the sclera when necessary; (2) irritation of the sclera and choroid in the region of the tear; (3) an attempt to reapply the retina against the choroid by evacuation of retroretinal fluid by intravitreous injection of air and by various techniques that tend to push the sclera and choroid toward the retina, by means of lamellar resections of the sclera, and the placing of small cylinders of plastic material fastened by mattress sutures.

The ophthalmologists of São Paulo can be proud to have organized a series of large congresses, that would do justice to any of the best developed centers in the world, in honor of the IV Centennial of the city. The large quantity of knowledge brought to light at these meetings will bear fruit for a long time to come for many papers of outstanding scientific quality were presented.

Jorge Balza.

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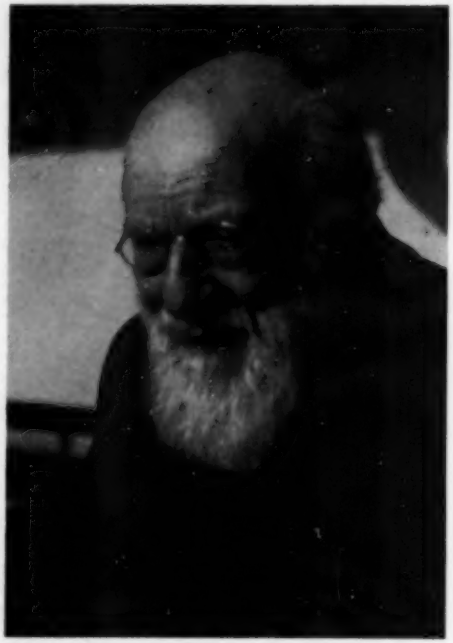
# OBITUARY

## MAXIMILIAN SALZMANN

## (1862-1954)

In Graz, Austria, on April 17, 1954, at the age of 91, the heart of Prof. Maximilian Salzmann stopped beating, a heart that had always been filled with compassion, kindness, and love for all mankind.

Professor Salzmann was born on December 10, 1862, in Vienna. He was graduated on March 12, 1887, from the University of Vienna and in the following year became an assistant of that illustrious ophthalmologist, Prof. Ernst Fuchs, at the University of Vienna. Salzmann was made a professor of ophthalmology in 1895. He resigned from the University of Vienna in 1911 to accept the chairmanship of the department of ophthalmology of the University of Graz. During his tenure at Graz he was asked by the governing body to return and assume the chairmanship of one of the two eye clinics at the University of Vienna. He, however, declined this high honor and decided to remain at the University of Graz where he retained the chairmanship of the department until 1932 when, having reached the age of 70 years, his retirement became mandatory.



PROF. MAXIMILIAN SALZMANN
(From a colored photograph taken by Professor Böck on Salzmann's 91st birthday)

Salzmann in addition to being one of the outstanding ophthalmologists of this century, was a gifted linguist and painter and had an authoritative knowledge of botany, geology, mathematics, and the arts. I was once informed that he could read and speak five languages fluently. Personal experience made

me acutely aware of his intimate and comprehensive knowledge of the more complex constructions of the English language. Salzmann's avocation was botany. His large herbarium, which was always at the disposal of those interested in this field, was a real showplace. When, in 1928, I once asked him if he would like to come to the United States, he said, "Yes, for one reason only." When I asked him what that reason might be, he replied, "To study the Yellowstone National Park."

Salzmann's lectures to his students were made crystal clear by the frequent resort to the blackboard where, with a few strokes of the chalk, he would quickly draw a detailed and exact reproduction of the normal or pathologic condition he was discussing. His paintings of normal and pathologic fundi were exact in all detail. He re-edited and enriched Jaeger's *Atlas of Diseases of the Fundi* by augmenting it with a number of his own fundus paintings. Another group of his original fundus paintings added an important supplement to certain editions of Fuchs' *Textbook of Ophthalmology*.

Above all, Salzmann was a gifted ophthalmologist, seeking perfection in all his work. His scientific achievements made him known and respected by ophthalmologists all over the world. Every subject in which he became especially interested, and they were many, was enriched by his endeavors. Salzmann was an authority of geometric optics and published a number of articles on various phases of this subject as it pertained to the normal, the myopic, and the aphakic eye, and to skiascopy. He analytically described the optics involved if one were to be able to see the sinus angle of the living eye. This was followed by devising, for the first time, a usable method of ophthalmoscopy of the sinus angle which subsequently proved to be the forerunner of present-day gonioscopy. His drawings of his observations of the sinus angle by indirect ophthalmoloscopy have not been surpassed in detail nor accuracy by the authorities of today, whose work in this field has been made so much easier by the use of the present-day armamentarium devised for the study of this part of the eyeball.

Salzmann published a number of histopathologic articles covering a wide variety of conditions, with special interest in glaucoma, myopia, and keratoconus. He contributed to several textbooks. In my opinion, his greatest contribution to ophthalmology was his book, *The Anatomy and Histology of the Human Eyeball in the Normal State: Its Development and Senescence*, which book developed as the result of the lectures Salzmann gave for many years on this subject to the students at the University of Vienna and to ophthalmologists of foreign lands who came to Vienna for postgraduate work. This scholarly book was translated into English by the late Dr. E. V. L. Brown and remains the reference work in this field.

Salzmann was an extraordinarily modest man and was always ready to help others in their scientific work, even if it cost him much in time and effort. He was always happy to place the facilities of his laboratory at the disposal of visiting ophthalmologists and would frequently suggest subjects, usually pathologic, and furnish the necessary material or stain slides for an article. After his retirement, Salzmann visited regularly his beloved clinic and, whenever asked, gave freely from his rich knowledge and tremendous experience. He always inspired those who came in contact with him.

For a number of years Salzmann was the victim of bilateral macular degeneration. As a result of this affliction, it was necessary for a member of his family to read to him and, because of his intense interest in ophthalmology, the readings were usually from the ophthalmic literature. It was also necessary for a member of his family to take dictation and type the articles which he submitted for publication, the last ones being published in the last years of his life.

This unusual man, who accomplished so

much, shunned publicity, lived simply and modestly in his garden-surrounded home on a hill in the city where he had worked for so many years. His spirit was filled only with the love for the sciences, the fine arts, and man.

Thus, an unusual career has come to an end. An amiable and inspiring, gentle and kind colleague of high scientific rank, a teacher always admired and revered, has been called to his final resting place.

Dewey Katz.

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## BOOK REVIEWS

ELEKTROCHIRURGIE AM AUGE. By Karl Safar. Vienna, Springer Verlag, 1953. 170 pages, 56 figures, several in color, author and subject indices. Price: $8.60.

Electrosurgery is the application of high-frequency current to surgical procedures. The heat engendered arises in the depth of the tissues, whereas the electrode remains cool. It is not to be confused with galvanocautery and other methods of cauterizing by the direct application of a heated mass of metal, nor with such other methods of using direct current as electrolysis.

After more than 20 years of experience in the use of electrosurgery in ophthalmology Professor Safar came to feel that a certain stability of opinion had been reached and that the time was ripe to write a book which would perspicuously exhibit the procedures, their great advantages, and their shortcomings, and would serve as a guide to ophthalmologists. Because of his extensive experience and because he has contributed considerably to the development of ophthalmic electrosurgery in every stage of its growth, he is eminently fitted for this task. He has succeeded admirably in writing a much-needed book which can be used as a reliable introduction to the subject.

The author's own contribution and his own experiences are described more extensively than information which he gathered from the literature. This makes for a livelier and more effective book and, since Safar's experience is so extensive and fruitful, his exposition gives an adequate and balanced account of the entire subject.

This treatise is a thoroughly practical book. The introductory discussion of the physical fundamentals is elaborated only so far as is necessary for an understanding of their application in actual surgery.

The field of application of ophthalmic electrosurgery is remarkably wide. In the lids this procedure is effective in many benign and inflammatory tumors and cysts. The procedure is particularly useful for removing the often refractory marginal chalazion. In the conjunctiva inflammatory follicles may often be removed most easily with diathermic stippling. Eversion of the lid may be treated by applying a globular electrode to the conjunctival surface.

The management of malignant neoplasms requires extensive knowledge and this is exhibited in the detailed case reports. The treatment of epibulbar growths is similarly clarified. Among intraocular abnormalities are staphyloma after limbal iris incarceration, filtrating scars after cataract extraction, and cysts in the anterior chamber.

In the vicinity of the lens great caution must be exercised to avoid injury to this structure but, for extraction of a dislocated lens, electrosurgery is occasionally extremely helpful. A needle which is completely insulated except for its point is introduced into the anterior chamber and its point is made to puncture the lens; when the current is closed, the lens is literally fried onto the needle with which it can be withdrawn from the eye with safety. Many uveal neoplasms may be effectively eradicated by electrosurgery, and the operations for the relief of glaucoma are well known.

Perhaps the most extensive and gratifying use of electrosurgery occurs in retinal detachment and almost 60 pages are devoted to the treatment of retinal disturbances.

A bibliography of over 1,000 references completes this very useful book.

F. H. Haessler.

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THE OPTOMETRIST'S HANDBOOK OF EYE DISEASES. By Joseph I. Pascal, M.D., and Harold G. Noyes, M.D., St. Louis, C. V. Mosby Company, 1954. Clothbound, 300 pages, 153 illustrations, bibliography, and index. Price: $9.50.

In this simplified account of eye diseases designed for the needs and interests of the optometrist, the treatment of the subject is naturally less technical than in handbooks for medical students though more detailed than presentations for the laity. An important purpose of the book is to prepare candidates for state optometric board examinations. In Chapter 25, 200 questions are listed, the answers to which are summarized in Appendix III. The following 10 examples give a fair idea of what the optometrist is expected to know:

Name five complaints of a patient who has glaucoma.

Name five causes of frontal headache not due to the eyes.

What refractive changes may result from interstitial keratitis?

Name five signs of thyrotoxicosis.

Name five causes of progressive myopia.

Name five causes of night blindness.

What pupillary changes point toward syphilis?

Give reasons why patent medicines should not be used.

Give four causes of secondary optic atrophy.

How may diabetes affect vision and refraction?

The problems of the near-blind are discussed and to what extent motivation can overcome the handicap of a poorly functioning eye is illustrated in the lives of Borghild Dahl and Aldous Huxley. According to the authors the reading slit was first described by C. F. Prentice in 1897 who called it a "typoscope."

The important epilogue quotes from the counsel for the American Optometric Association:

"An optometrist is concerned solely with the correction and improvement of vision and the accomplishment thereof without the use of drugs, medicine, or surgery. . . . One is not obliged to offer or render first aid. The removal of a foreign body from the eye is always fraught with attendant risk. A good standard rule to follow is that, if the speck is embedded in the cornea, the optometrist under no circumstance should attempt to remove it, but should refer the patient for medical care."

James E. Lebensohn.

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OPHTHALMIC SURVEY OF THE KIMBERLEY DIVISION OF WESTERN AUSTRALIA. By Ida Mann. Perth, Western Australia Health Department, 1954. 43 pages, 3 maps, 17 photographs, 17 tables.

This important publication presents in detail the results of an ophthalmic survey of a sparsely inhabited part of tropical Australia. "The Kimberleys" is a vast area of cattle and sheep stations some of them millions of acres in extent. The project necessitated 9,700 miles of travel and nearly three months in time. The report is divided into eight parts plus three appendices.

The itinerary is described. Equipment was taken for examination of eyes, for keeping records, for the treatment of ophthalmic and other emergencies, and for simple pathologic procedures. The procedure adopted in conducting the survey is described, as are the localities visited. A total of 2,866 subjects were examined of whom 679 were white, 507 colored, and 1,680 aborigines; 41.84 percent of the total number examined had trachoma. The largest cause of monocular blindness (111 cases) was injury. The largest cause of binocular blindness (149

cases) was trachoma. The incidence of cataract as a cause of blindness is definitely much lower than in Asia and is similar to that in England. Glaucoma is uncommon and severe myopic degeneration was not encountered. Apart from trachoma the eyes are generally healthy.

Types of eyes and normal variations, including color vision and visual acuity, in white and colored subjects and in aborigines are described. Detailed consideration of the diseases found includes several tables concerning the distribution of trachoma. Conditions of dirt, squalor, and personal disregard for hygiene accompanied the high trachroma rate. Bush flies were thought to be the most common vector. Pteryguim showed no racial differences. Various types of conjunctivitis were encountered, some occurred in outbreaks. Phlyctenular keratitis is very rare. Ocular leprosy was seen, especially at the leprosarium.

There are notes on the various localities visited. The author emphasizes the necessity of raising the standard of living but this will be difficult.

Appendix I, on "Trachoma and other forms of conjunctivitis," is a leaflet sent by the Department of Public Health to any interested person. Appendix II, "Notes on the control of eye disease in a leprosarium" is circulated to the staff of leper hospitals, and Appendix III, "Abstract from the official records of the northern territory medical services," gives the results of a smaller ophthalmic survey.

Ronald Lowe.

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OBSERVATIONS ON HOLE IN THE MACULA. By Paul Tower. Basel, S. Karger, 1954. 55 pages, 15 figures, references, no index. Price: $2.00.

The author, in this short monograph, has reviewed for us the subject of the hole in the macula, a subject that has bothered ophthalmologists for 80 years, and is still not completely recognized nor universally understood. He points out the frequency with which holes in the macula are encountered and that they are no longer considered to be exclusively traumatic in origin, but are more often the end result of various, ordinarily unrelated, pathologic processes that cannot be coordinated with specific etiology.

Part 1 consists of a historical review and a discussion of etiology. Part 2 is made up of a description of the pathologic processes (mostly edema and cystoid degeneration of the retina) and concludes with a neat pathologic classification. Part 3 discusses the ophthalmoscopic features (including the use of the Hruby lens) and the therapy (mostly empirical) and the prognosis.

Derrick Vail.

# ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

## CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

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## 1

### ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Badtke, G. **Abnormal development of the human lens.** Arch. f. Ophth. **155**:17-25, 1954.

A lens from a five-months-old human fetus is described in which the capsular epithelium occupied the posterior surface of the lens. Both lenses of the fetus showed signs of subcapsular cataract. (3 figures, 17 references) Ernst Schmerl.

Morone, G., and Sacchi, S. **Histochemical study of the eyelids.** Rassegna ital. d'ottal. **23**:154-160, March-April, 1954.

Knowledge of the histochemistry of the skin and glands of the eyelids is relatively limited. The authors studied the desmona of Schaffer or the corpuscles of Ranvier-Bizzozzero, these being the corpuscles distributed among the structural fibers, and the cells described by Wolff, considered as unicellular sebaceous glands. The former are identified only with great difficulty. The cells of Wolff give a positive reaction to Sudan black B and are generally found in groups. The absence of fluorescence is noted in the fatty granules of the xanthoma, but was present in the desmona cells. (5 figures, 7 references) Eugene M. Blake.

## 2

### GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Aterman, K., and Greenberg, S. M. **Experimental exophthalmos produced by cortisone in rats.** A.M.A. Arch. Ophth. **51**: 822-831, June, 1954.

Experiments with albino rats showed that exophthalmos could be regularly produced by the administration of cortisone. Cortisone did not have this effect in hypophysectomized rats. A severe hyperthyroid state also inhibited the exophthalmic action of cortisone. The authors suggest that the thyroid hormone produces this effect on ocular protrusion by presenting the building up of an excess of adrenocortical steroids. (2 figures, 3 tables, 15 references) G. S. Tyner.

Fontana, Guiseppe. **Implantation of adenomcarcinoma in anterior chamber of rabbit.** Rassegna ital. d'ottal. **23**:177-188, May-June, 1954.

Following the method described and

developed by Harry Green the author repeated these experiments. He reviews the results obtained in attempts to graft the adeno-carcinoma of Ehrlich into the anterior chamber of the rabbit's eye. He stresses the constant lack of success and then reviews the literature. He re-affirms the utility of the principles of general oncology, wherein it is established that it is not possible, under any conditions, to obtain growth of implanted heterogeneous neoplastic tissue. (43 references)

Eugene M. Blake.

Kirber, H. P., and Kirber, M. W. **Experimental intraocular infection with mumps virus.** A.M.A. Arch. Ophth. **51**: 832-849, June, 1954.

Ocular lesions appeared in the guinea pig after inoculation of living and attenuated mumps virus into the anterior chamber, cornea, subconjunctival space and skin. The serological response of the animals to the various mumps antigens was studied. Some of the corneal changes seen clinically in man were noted in this experimental series. The corneal stroma was uniformly involved except for a clear peripheral zone. The recovery time from keratitis was much less in guinea pigs than man. In the animals there was a uniform transient involvement of the anterior portion of the uvea. Ocular lesions were produced only by living virus and no multiplication of the virus in the inoculated eye could be demonstrated. The virus could not be passed from an inoculated eye to a normal eye. (14 figures, 25 references)

G. S. Tyner.

Mitzukawa, T., Kishimoto, S., and Kizu, S. **Ocular allergic reaction to lens protein.** Acta Soc. Ophth. Japan **58**:762-767, Aug., 1954.

Water-soluble and insoluble protein of the crystalline lens were used as antigen. Rabbits were sensitized with these proteins. A passive sensitization was attempted by Landsteiner's method (Proc. Soc. Exp. Biol. Med., 49:688, 1952) by a transmission of peritoneal cells to other rabbits. When the antigen was introduced into the vitreous of these rabbits a severe reaction was brought about when insoluble proteins were used as antigen. When a passive anaphylaxis was brought about by a transmission of the serum, a severe reaction was observed only by soluble protein.

In actively sensitized animals, an intracorneal reaction was positive when insoluble protein was used but not with soluble protein. A cutaneous reaction was, however, positive with both these proteins. The authors conclude that the allergic reaction with soluble lens protein is an Arthus type and with insoluble protein there is a tuberculin type of reaction. (1 figure, 16 references) Yukihiko Mitsui.

Shimoyama, J. **Ocular manifestation of experimental endocarditis.** Acta Soc. Ophth. Japan **58**:648-650, July, 1954.

Streptococcus sanguinis and antiserum against the valve of rabbit's heart were introduced into the rabbit intravenously and an endocarditis was produced in the rabbit. In three of the 20 eyes examined, small hemorrhages were seen in the retina with an ophthalmoscope. The biopsy finding was, however, obvious in the equator of the eye, particularly in the uvea. The finding was hemorrhage, leucocytic infiltration and venous congestion.

Yukihiko Mitsui.

Suie, T., and Sudimack, J., Jr. **Capillary tube method for antibody determination.** A.M.A. Arch. Ophth. **51**:875-877, June, 1954.

The authors describe a method whereby small quantities of aqueous humor may be titrated against a series of antigens. Its practical application lies in the diagnostic

study of non-granulomatous uveitis. The capillary tubes were made of soft glass, 0.8 by 90.0 mm. The antigen was drawn up to approximately 6.0 mm., and an equal amount of aqueous was allowed to flow into the same end. Caution was exercised to prevent the entry of air bubbles. The tubes were inverted and inserted at a 45-degree angle into Plasticine. The inversion permitted the union of the antigen and aqueous. Controls for antibody and antigen were included. All tubes were incubated at 37 C. for three or eight hours. The tests were read against a white background under a strong light with the aid of a magnifying glass. (2 tables, 2 references) G. S. Tyner.

## 3

### VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Cucco, G. **The action of organ-specific lipoids on experimental retinopathy caused by sodium iodate.** Ann. di ottal. e clin. ocul. **80**:101-112, 1954.

In 20 rabbits an experimental retinopathy was provoked by daily injections of 1.5 cc. of a 4.5 percent solution of sodium iodate. Ten animals were used as controls, the other ten were given injections of "Cellulin Retinale," a prepared extract of animal retinas. All control animals exhibited characteristic lesions. The treated animals also showed some disturbance but of a much milder degree, both ophthalmoscopically and histologically.

John J. Stern.

D'Asaro, B. S., Young, R. G., and Williams, H. H. **Biochemistry of the eye. III. Lipids of the lens, cornea, iris, ciliary body, and retina.** A.M.A. Arch. Ophth. **51**:596-598, May, 1954.

The various parts of fresh bovine eyes were dissected and separated, ground and frozen. Partial fractionation of the lipids in lens, cornea, retina, iris and ciliary body revealed that most of the fatty acid is bound as "essential lipid." The wide quantitative differences in lipid content of the various structures implies a wide range in metabolic activity. The vitreous and lens are relatively inert.

G. S. Tyner.

Esser, H., Heinzler, F., and Pau H. **Electrophoretic separation of the protein fractions of the human aqueous.** Arch. f. Ophth. **155**:11-16, 1954.

The authors present rather similar electrophoresis curves for aqueous and cerebrospinal fluid. Both curves differ significantly from the curve for normal blood serum. The $\gamma$-globulins of the aqueous are considerably increased under pathologic conditions. (8 figures, 2 tables, 9 references) Ernst Schmerl.

Gandolfi, A. **Experimental investigations on the re-epithelializing effect on the cornea of chlorophyll, chloramphenicol and codliver oil, alone and in combination.** Ann. di ottal. e clin. ocul. **80**:131-142, 1954.

In 40 adult rabbits the cornea was completely denuded of epithelium, and the re-epithelialization observed under the influence of 1 percent chlorophyll, 10 percent codliver oil, and 1 percent chloramphenicol, alone or in several combinations, in an ointment base. Chlorophyll had the most powerfully stimulating effect. Chloramphenicol slightly retarded the epithelial repair, an effect which could be counteracted by the addition of codliver oil.

John J. Stern.

Garner, L. L., Carl, E. F., and Grossman, E. E. **Effect of cigarette smoking on flicker fusion threshold.** A.M.A. Arch. Ophth. **51**:642-655, May, 1954.

There is apparently a wide variation in individual response to cigarette smoke. Among 108 subjects tested, cigarettes raised the flicker fusion frequency in 35

and lowered it in 21. The consistency of the data was limited by the imperfections of the apparatus and technique employed. The lowering of flicker fusion frequency is apparently due to anoxia and could be raised by administration of nitroglycerine.

G. S. Tyner.

Gemolotto, Guglielmo. **Further research on the content of glucose and ascorbic acid in the aqueous and on the electrophoretic changes in the lens of rabbits with experimental choroidoretinal degeneration.** Gior. ital. oftal. 7:75-79, Jan.-Feb., 1954.

In previous studies the author had shown that during the phase of degeneration there is an increase of glucose in the aqeous of rabbits and a decrease of ascorbic acid; he now has demonstrated that after a lapse of 15 months there is a tendency for the glucose and ascorbic acid to return to their normal quantities. In the same animals the lens showed an inversion of the normal relative quantities of alpha and beta soluble proteins, with a decrease in the quantity of the latter; such a change had been found by others in cataractous lenses. (2 figures, 14 references) V. Tabone.

Giardini, A., and Bagolini, B. **Influence of sodium pentobarbital (nembutal) on the permeability of the blood-aqueous barrier to fluorescein.** Ann. di ottal. e clin. ocul. **80**:25-30, 1954.

Eight rabbits were used, and in seven fluorescein appeared in the anterior chamber 2 to 3 minutes earlier under nembutal narcosis than in normal controls. In five animals the concentration of fluorescein rose constantly while the experiment was in progress. It is concluded that nembutal causes a disturbance of the blood-aqueous barrier, usually in the direction of increased permeability. John J. Stern.

Giardini, A., and Gandolfi, A. **Investigations of the behavior of the thickness of the cornea after perilimbic thermocauterization and diathermocoagulation.** Ann. di ottal. e clin. ocul. **80**:43-54, 1954.

The corneal thickness was used as an index of the water-content of the cornea after thermocauterization and diathermycoagulation of the limbus in 18 rabbit eyes. Between the third and the twelfth hour after the beginning of the experiment a diminution of the corneal thickness up to 22.2 percent was observed. This was followed by a secondary increase in thickness. The decrease of the thickness of the corneal tissue is due to a loss of fluid into the aqueous and the conjunctival sac, either because of a difference in tonicity, or an active participation of the endothelium and epithelium.

John J. Stern.

Hörmann, Emmerich. **The pathogenesis of the galactose cataract of the white rat.** Arch. f. Ophth. **154**:561-573, 1954.

The following questions were studied: 1. how does the white rat fare when fed a diet containing different concentrations of galactose? 2. which concentrations produce cataracts? and 3. can the action of galactose be controlled? Ten series of five young rats each were used and fed a diet described by Yudkin and Arnold (Arch. Ophth. 14:960, 1935). It was found that a rat which is fed 70 percent of α- or β-lactose soon dies from inanition; one fed on 30 percent galactose and 40 percent oatmeal develops lens opacities. Injections of the enzyme cocarboxylase inhibit the actions of lactose as well as of galactose. (87 references) Ernst Schmerl.

Noda, T. **A histochemical study of phosphatase in the rabbit eye.** Acta Soc. Ophth. Japan **58**:651-666, July, 1954.

This is a study carried out by a modified procedure originally recommended by Gomori. The following portions are

strongly positive for alkaline-phosphatase reaction: namely, the superficial layer of the corneal epithelium, the ciliary processes, the outer nuclear layer of the retina, and the choroid. The corneal endothelium is positive for the reaction, although only slightly, in contrast to the negative result of previous investigators. The author further describes the change in the content of this ferment by infection and injury of the cornea. He finally concludes that the phosphatase content in the corneal endothelium increases and decreases with an increase and decrease in metabolism in the endothelium. (16 figures, 18 tables, 29 references) Yukihiko Mitsui.

Pau, Hans. **The physiology and pathology of the cornea.** Arch. f. Ophth. **154**: 579-602, 1954.

The author studied changes of corneal permeability and transparency produced by metabolic disurbances of the epithelium and endothelium. Calf and beef corneas were used. Normal epithelium and endothelium seem to give the best protection against swelling and clouding. Bowman's and Descemet's membrane protect to a slightly less degree. The breakdown of the normal metabolism might be the main cause of the pathologic swelling of the cornea. Urethane, for instance, damages the corneal metabolism and at the same time accelerates corneal clouding and swelling. The colloid osmotic pressure of corneal and scleral proteins acting upon aqueous and tears is said to amount to 1000 mm. of water. The site of enzymes within the cornea might play a role in active transfer of ions. Dehydrases seem to occur chiefly in the deeper layers of the epithelium and in the endothelium. Damage to the enzyme systems and the corneal metabolism is considered the main factor in changes of corneal permeability. (7 figures, 59 references) Ernst Schmerl.

Rizzini, Vittorio. **Action of Mintacol as an antagonist of mydriatic drugs.** Gior. ital. oftal. **7**:80-82, Jan.-Feb., 1954.

Mintacol (diethil-p-nitrophenylphosphate-E 600, Bayer) proved very effective in counteracting the effect of mydriatic drugs, including atropine and hyoscine. The author made some observations on the comparative action of pilocarpine, eserine, and DFP Mintacol, which was used in an aqueous concentration of 1/6000 and had a less lasting action than atropine. (1 figure) V. Tabone.

de Roetth, A., and Orchen, M. **Glycolytic activity of ciliary processes.** A.M.A. Arch. Ophth. **51**:599-608, May, 1954.

From a study of material obtained from 600 beef eyes, the author concludes that the glycolytic activity of the ciliary epithelium is in the same order of magnitude as that of the retina, thus placing it among the most active of mammalian tissues. G. S. Tyner.

Sakanoue, T. **Experimental study on the blood pressure of the central vein of the retina.** Acta Soc. Ophth. Japan **58**:614-626, July, 1954.

In the first part, Sakanoue describes the design of an apparatus and a method of measuring the blood pressure in the central vein of the retina. In normal man the blood pressure of the central vein was found to be 0.5 to 1.5 mm. Hg higher than the intraocular pressure. In the following parts he describes the change in the venous pressure after a compression of the neck. When the neck is compressed by 100 mm. $H_2O$ or more on the same side or by 150 mm. $H_2O$ on the opposite side, an increase in the blood pressure in the retinal vein occurs about 10 seconds later. The author considers the increase in the venous pressure in the retina by the compression of the opposite side of the neck to be secondary to an increase in the

arterial pressure on the same side, while a compression of the side directly causes a venous stagnation on the same side. (6 figures, 15 tables, 29 references)

Yukihiko Mitsui.

Sarwar, M. **Some observations on the physiology of the cornea.** Tr. Ophth. Soc. U. Kingdom **73**:547-553, 1953.

The author reviews opinions on the process of the maintenance of corneal transparency to facilitate an understanding of the problems of the corneal haze which occurs in wearers of contact glasses. He is convinced that the loss of corneal transparency is due to an intake of fluid by the cornea. The appearances and symptoms vary with the amount of fluid absorbed. In these eyes there was a loss of corneal sensitivity. The cornea is generously supplied with unmyelinated fibers and it would seem probable that where there is continued stimulation of these fibers, such as probably occurs in subclinical errors of fit, there is a migration of sodium and potassium ions from the interstices of the corneal substance. This ionization results in a rise of the osmotic pressure inside the cornea with a consequent inflow of water into it.

Beulah Cushman.

Sbordone, Girolamo. **Modifications of lens transparency following experimental paracentesis of the cornea.** Rassegna ital. d'ottal. **23**:161-170, March-April, 1954.

Repeated paracentesis of the rabbit cornea demonstrated marked changes in the composition of the secondary aqueous as compared to the primary. The following changes were noted in the secondary fluid: 1. specific weight, osmotic pressure and viscosity increased, 2. electric conductivity diminished, 3. protein content increased, 4. amount of enzyme increased in proportion to the increase in proteins, 5. anions decreased, cations increased, 6. glucose, urea and other electrolites generally increased, 7. the pH was unchanged, 8. the alkaline reserve was unchanged, and 9. pyruvic and lactic acid increased. Repeated paracentesis was followed by opacification of the superficial, and, at times, of the deeper layers of the lens. Apparently some substance, or substances induced an increase in the permeability of the lens capsule. (4 figures, 53 references) Eugene M. Blake

Schlegal, H. E., and Swan, K. C. **Benoxinate (Dorsacaine) for rapid corneal anesthesia.** A.M.A. Arch. Ophth. **51**:663-670, May, 1954.

A new topical anesthetic agent is described which after clinical and laboratory appraisal appears to be a safe and reliable drug. G. S. Tyner.

Schwab, F. **Experiments with ultrasonic impulses upon the eye.** Arch. f. Ophth. **155**:97-114, 1954.

The author studied the effect of ultrasonic waves upon lenses of enucleated hogs' eyes. He used an apparatus which produced continuous and intermittent ultrasonic waves having a frequency of 1,000 and 5,000 kilohertz. Varying his experimental arrangements, the author found that within a few minutes cortical as well as nuclear cataracts formed. He ascribes the cortical types of cataract chiefly to the mechanical effects, and the nuclear cataracts to the thermal effects of the ultrasonic waves. (5 figures, 9 references) Ernst Schmerl.

Siliato, F. **The action of adenosin-triphosphoric acid (ATP) on the permeability of the blood-aqueous barrier.** Ann. di ottal. e clin. ocul. **80**:60-74, 1954.

The effect of ATP on the permeability of the blood-aqueous barrier was estimated in rabbits after subconjunctival and retrobulbar injections of the substance. The method of Amsler and Huber was used for the measurements. An increased capillary permeability in the eye was

found after administration of the drug, the highest values being obtained after retrobulbar injections. The author assumes that the site of action of the drug is in the posterior ciliary arteries.

John J. Stern.

Simonelli, M., and Rizzini, V. **Isonicotinic hydrazide in ophthalmology.** Gior. ital. oftal. **7**:199-202, May-June, 1954.

The effect of isonicotinic hydrazide in 40 proved cases, and in 20 presumed cases of ocular tuberculosis was very favorable. The best results were obtained in cases of iridocyclitis and in those of choroiditis. (5 references) V. Tabone.

Stepanik, J., and Kemper, R. A. **Outflow of aqueous humor.** A.M.A. Arch. Ophth. **51**:371-380, May, 1954.

The authors attempted to determine quantitatively to what extent the clear or almost clear aqueous veins participate in the transport of aqueous. They estimate that the visible outflow ranged from 6.51 to 69.02 percent of the total aqueous outflow. The remainder of aqueous is apparently already mixed with blood and cannot be observed. The total aqueous outflow was determined by Grant's tonographic method. The visible flow was calculated by determining the current velocity and measuring the caliber of the veins. The current velocity was determined by touching an aqueous vein upstream and forcing a small group of blood cells to flow through the vein. The velocity of these cells could be measured.

G. S. Tyner.

## 4

### PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Campbell, F. W., and Primose, J. A. E. **The state of accommodation of the human eye in darkness.** Tr. Ophth. Soc. U. Kingdom **73**:353-361, 1953.

The authors discuss the production of night myopia and describe experiments carried out which show that spherical and chromatic aberrations and also a change of accommodation of the eye play a part in bringing it about. There are difficulties in measuring accommodation in darkness. Constant light is required for skiascopy and for coincidence optometry. However, it is possible to determine the shape of the lens from the size of the Purkinji-Sanson image which can be photographed if the light is flashed only at intervals.

Beulah Cushman.

Dykerhoff, Dirk, and Siebeck, Robert. **Measurements of the stereoscopic image produced by haploscopic pictures of equal and unequal brightness.** Arch. f. Ophth. **154**:637-655, 1954.

The authors used a special haploscopic arrangement to study the problem described in the title of their paper. They found that haploscopic pictures of equal brightness produced a virtual image of less depth than was to be expected from trigonometric calculations. The frontoparallel position of the haploscopic pictures seems to some extent to counteract the depth impression due to the binocular parallaxis. Diminishing brightness of one of the haploscopic pictures exercises its influence upon the fused image with respect to lateral and depth inclination. For further details the original paper must be consulted, and also for comparison with Verhoeff's studies (A.M.A. Arch. Ophth. 10:640, 1933 and 13:151, 1935). (6 figures, 8 tables, 37 references) Ernst Schmerl.

Givner, Isadore. **Visual loss following cardiac arrest.** A.M.A. Arch. Ophth. **51**: 787-879, June, 1954.

The visual loss following cardiac arrest is apparently due to cortical rather than retinal damage. (5 references)

G. S. Tyner.

Jean-Gallois. **Various causes of pro-**

**voked hypervision.** Bull. Soc. d'ophtal. France **2**:195-198, Feb., 1954.

The author has been interested in the photo-vasodilator reflexes for a long time. He points out the fundamental pattern of his experiments, namely, the relationship of monocular illumination, vasodilatation in the fundus, increased visual acuity, and decrease in ocular tension. He further discusses the possibility that corneal anesthetics may cause increases in visual acuity. Alice R. Deutsch.

Manas, L., and Shulman, P. **The variation in the accommodative-convergence accommodation (ACA) ratio upon periodic retesting.** Am. J. Optometry. **31**:385-396, Aug., 1954.

Most writers on the subject of the accommodation-convergence ratio have been impressed by the constancy of the measurement when taken over a course of years. Some claim that even the changes with presbyopia do not change the basic reflex unit. Now, repeated measurements on a group of 22 college students show a variation from time to time amounting to as much as 1.5 prism diopters per diopter of accommodation enforced. It will be necessary for others to repeat their studies on the effect of orthoptic training or of age.

Paul W. Miles.

Miles, Paul W. **Optics and visual physiology.** A.M.A. Arch. Ophth. **51**:880-902, June, 1954.

The literature for 1953 is abstracted and reviewed. (223 references) G. S. Tyner.

Neill, J. C. **A second report on the fitting and use of microlenses.** Am. J. Optometry. **31**:411-415, Aug., 1954.

Practical and mechanical aspects of the fitting of corneal contact lenses are discussed. If the lens moves too freely with eye movements, the corneal surface must be ground to a shorter radius. They are not satisfactory for keratoconus. Fitting can be made by inspection with fluorescein, but it is better to know the keratometer readings of the cornea.

Paul W. Miles.

Ridley, Frederick. **Clinical survey of 600 N.H.S. contact lens cases.** Tr. Ophth. Soc. U. Kingdom **73**:73-86, 1953.

The author discusses the result of 600 patients who applied for contact lenses. Of these 130 were rejected at the interview, 221 were not completed and 221 were finally fitted with contact lenses. Of the completed 221, 28 percent (60 patients) wear them all day, 72 percent (158 patients) have a standard wearing time of 16 hours a day, that is they wear them all day but take them out once or twice a day, if necessary, because of mucous and epithelial debris which collects under them.

Patients with myopia made up 43 percent of the patients and they wore the lenses an average of 12 hours a day. Patients with myopia greater than 15D derived visual advantage because the visual field was greatly extended. The patients with unilateral aphakia, or 16 percent of the patients, and patients with corneal nebula (10 percent) had a standard wearing time of 14 hours. Patients with high astigmatism were 5 percent of the group, those with keratoconus 4.5 percent and patients with corneal grafts 3 percent. Of the latter 19 patients, five improved from counting fingers to 6/60, 6/36, 6/18 and 6/61, one from 6/60 to 6/9; of three from 6/36, one improved to 6/6, and two to 6/9. Patients with mustard gas keratitis (2.5 percent) wear the contact glasses with tolerance until the associated chest condition deteriorates.

Beulah Cushman.

Rzymkowsky, J. **Stereophotographic and stereophotogrametric reproduction of the cornea and sclera of the living eye.**

Am. J. Optometry **31**:416-422, Aug., 1954.

A new method of determining the shape of the rear surface of a contact lens by photographing the globe is described. It is claimed that a better scleral fit can be obtained because the eye is comfortable and steady for the moment required. With the impression method, lid pressure or eye movements may spoil the mold. (10 figures) Paul W. Miles.

Umetani, S. **Influence of the size of the test object on the time of accommodation.** Acta Soc. Ophth. Japan **58**:757-774, Aug., 1954.

The time of accommodation, from infinity to 15 cm. in front of the eye was measured under various conditions. When a larger test object, printed with the Landolt ring of the same size is used under a constant illumination, the time of accommodation becomes shorter. The following formula is applicable if the diameter of the object is in the range of 2 to 32 mm.: $Y = a \log x + b$, where Y is the area of the subject, x is the time of accommodation and a and b are constant. With increased illumination the time of accommodation becomes shorter, even when the same object is used. However, the maximum is 1,000 lux. Over 1,000 lux, the situation becomes reversed, probably due to a glare. When the pupil is made small by an artificial pupil, the time becomes shorter, probably due to an increased depth of the focus. (7 figures, 4 tables, 13 references) Yukihiko Mitsui.

Weale, R. A. **Some aspects of total color blindness.** Tr. Ophth. Soc. U. Kingdom **73**:241-249, 1953.

Cone-monochromatism, which is recognized solely by the subject's inability to distinguish with photopic vision between two colors of equal luminescence, all other clues being absent, is a type which is of paramount interest to students of color vision. Its incidence has been estimated at about one in 100,000,000. A large number of people responded to two appeals—in 1950 and 1952—in the Sunday Express. Although color-naming tests and Ishihara tables were used, no conclusive result was possible without a color-matching test. For this purpose a chromatoscope was constructed whose design is described in detail. With its help five cone-monochromats were identified and three of them were subjected to further tests. It was with these three that Fincham made one of the most dramatic discoveries of the decade about human color vision. In each subject the accommodation reflex was more sensitive to the red-green mixture than to the monochromatic yellow which gave rise to the same sensation. The cone-monochromats reacted like color-normal subjects in this respect, but unlike red-green confusors. Beulah Cushman.

## 5

## DIAGNOSIS AND THERAPY

Esente, I. **Cortisone in lesions of the optic nerve due to tuberculous meningitis.** Gior. ital. oftal. **7**:212-224, May-June, 1954.

Cortisone was used in 29 cases of amblyopia during the course of tuberculous meningitis. Slight improvement in the peripheral fields was noted in some of the patients. The drug proved useless in miliary tuberculosis of the choroid. It is suggested that cortisone be used also in amblyopia due to papilledema and in hydrocephalus, because of its antitoxic and fibrinolytic action. (14 references) V. Tabone.

Lepri, Giuseppe. **Cortisone in clinical ocular tuberculosis.** Gior. ital. oftal. **7**:149-168, March-April, 1954.

The author sent a questionnaire on the action of cortisone in ocular tuberculosis to ophthalmologists all over the world, and from answers received and his own experience he draws the following conclusions: 1. Cortisone should not be given

systemically, but applied locally. 2. It can be safely used in all cases of ocular tuberculosis, and is especially valuable in lesions of the anterior segment. Specific anti-tuberculous therapy should be instituted at the same time. 3. Cortisone does not diminish and sometimes it enhances the effect of anti-tuberculous drugs. 4. Cortisone is less useful in lesions of the posterior segment. (24 references)

V. Tabone.

Sherman, R. A. **Cinerama, cinemascope and 3-D.** Am. J. Optometry **31**:397-410, Aug., 1954.

This paper illustrates cameras and special equipment used for taking stereoscopic and Cinerama movies. The latter can be appreciated by persons unable to fuse binocular images, while the former requires stereopsis. Stereoscopic methods are to be preferred because of the excellent sense of depth, but also because it calls attention of the public to binocular skills previously ignored. In some cases, movies would be ideal for purposes of visual training. Paul W. Miles.

## 6

## OCULAR MOTILITY

Alajmo, Arnaldo. **Retraction syndrome.** Gior. ital. oftal. **7**:141-148. March-April, 1954

In a case of unilateral retraction of the globe the findings were 1. enophthalmos, ptosis, and divergent strabismus in the primary positions; 2. reduction of adduction and of the oblique movements of the eye; 3. during adduction, accentuation of retraction and of ptosis; 4. absence of vertical movements; 5. absence of convergence; 6. anomalies of the iris; and 7. rotatory nystagmus. This case cannot be included under any of the three categories described by Malbran. A fourth category, for cases with the above findings may be needed. The possible causes for the syndrome are discussed. (5 figures, 5 references) V. Tabone.

Franceschetti, A., Monnier, M., and Dieterle, P. **Electro-oculographic analysis of oculomotor disturbances. (EOG).** Schweiz. Akad. Med. Wissen **10**:124-134, June, 1954.

The electro-oculography (EOG) provides an objective method for demonstrating oculo-motor disorders. The authors have shown that concomitant strabismus is a simple anomaly of position and that the EOG does not show pathological amplitude when the squinting eye departs from the primary position. In a case of paralysis of the lateral rectus muscle with retraction of the globe (syndrome of Stilling-Tuerk-Duane), the EOG revealed a diminution of amplitude in the direction of the antagonist of the paralysed muscle, which cannot be explained except by a slowing of adduction movements. On the other hand, in a case of paralysis of gaze to the left, with paralysis of the left medial rectus, the abduction of the right eye produced an exaggerated electro-oculographic amplitude, whereas the amplitude to the return movements was diminished. (5 figures, 7 references)

F. H. Haessler.

François, J. **Surgical treatment of ocular paralyses.** Gior. ital. oftal. **7**:89-100, March-April, 1954.

Five cases of convergent strabismus due to paralysis of the external rectus muscle are described. Treatment consisted in the transplantation of strips from the tendons of the superior and inferior recti and, in four of the cases, resection of the external rectus. Results were satisfactory. (10 figures, 11 references) V. Tabone.

Goto, N. **A study of optic nystagmus by the electro-oculogram.** Acta Soc. Ophth. Japan **58**:851-865, Aug., 1954.

This interesting study is similar to that

of Anderson (Brit. J. Ophth. *37*:*267*, 1953) performed independently. Goto studied the electro-oculogram in cases of congenital nystagmus and subdivided the nystagmus into true optic nystagmus and a nystagmus due to an abnormal position of rest. The latter corresponds to the eccentric nystagmus of Anderson. Goto describes the characteristics of the eccentric nystagmus in detail. The visual acuity is usually over 20/70, there is a characteristic head posture indicating an eccentric rest position, and ocular movement is apt to be more frequent than 150 cycles per minute. The movement is concomitant and is horizontal and rhythmic. Four cases of such nystagmus were operated upon. An advancement of the rectus muscles of both eyes was performed in order to recover the normal rest position. There is a considerable improvement in the vision, nystagmus and in other symptoms. In three of four patients a vision of 20/20 was restored. Instead of Anderson's retroplacement, the author uses the advancement technique. In the analysis of the electro-oculogram, he recognized a lowering of the tonus in the internal and the external rectus muscles. Therefore, he avoided a retroplacement. (6 figures, 3 tables, 12 references) Yukihiko Mitsui.

Kuboki, K. **Electromyogram of human ocular muscles.** Acta Soc. Ophth. Japan **58**:874-877, Aug., 1954.

This is an introduction of a procedure to obtain electromyograms of the ocular muscles in man. An interference voltage and a discharge from a neuromuscular unit are illustrated. The latter discharge is one-, two- or three-phasic, with an amplitude less than 200 micro-volt, having 10 to 70 cycles. (5 figures)

Yukihiko Mitsui.

Lyle, T. Keith. **(Doyne Memorial Lecture) Factors which affect the prognosis and treatment of ocular palsy.** Tr. Ophth. Soc. U. Kingdom **73**:435-502, 1953.

The subject of this extensive discussion is based on 135 cases of congenital ocular palsy. 86 patients had palsy with constant strabismus and (in all but four) with diplopia. Of the patients with congenital palsy 86 patients had diplopia and of these 37 with suppression. The superior rectus muscle was involved 59 times in the first group, and eight times in the second group. The superior oblique muscle was defective 39 times, the lateral rectus 17 times (3 bilateral), the inferior oblique 6 times, and inferior rectus 5 times. There were two patients with congenital partial third nerve palsy in which all the muscles supplied by the third nerve of one eye were affected. The author believes that surgery is indicated for the most rapid and complete resumption of normal activities and to prevent development of deformities and perversions of central nervous control. (38 figures, 4 tables)

Beulah Cushman.

Quereau, J. V. D. **Some aspects of torsion.** A.M.A. Arch. Ophth. **51**:783-788, June, 1954.

The purpose of this paper is to help clarify the distinction between true and false torsion. False torsion is described and a method of observing real torsion objectively is presented. The fixed amount of tilt of the vertical meridian of the cornea which accompanies oblique movements of the eye from a primary to a teritary position is false torsion. The vertical meridian is inclined slightly toward the original primary position. This amount of torsion accompanies a similar movement of any sphere. False torsion is detected objectively only when the eye is viewed against the conventional planar coordinate system. When the movement of the globe is sighted along an oblique axis of the direction of movement false torsion is not observed. Any torsion noted

in this manner is true torsion. (9 figures, 9 references) G. S. Tyner.

Reed, Howard. **Treatment of paralysis of the extraocular muscles.** Canad. M.A.J. **70**:628-631, June, 1954.

Paralyses due to multiple sclerosis or a vascular lesion almost always recover without surgery. In congenital cases without binocular vision and in patients over eight years of age, cosmetic surgery is indicated. Patients with traumatic lesions should be examined with the Maddox rod and Hess screen at intervals of three weeks. When the findings are unchanged after two successive intervals, surgery is indicated. (1 figure, 1 table, 1 reference) Irwin E. Gaynon.

## 7

## CONJUNCTIVA, CORNEA, SCLERA

Ching, Renald. **A new trachoma concept.** A.M.A. Arch. Ophth. **51**:750-761, June, 1954.

"Trachoma is a mild, insidious disease, frequently self-limiting, not especially infectious, comparatively harmless. . . ." The author thus defines, "true trachoma," due to infection with a filterable virus. "Infected trachroma," the result of one or a variety of secondary organisms in addition to the trachroma virus, is the entity commonly described in standard texts. Criteria of diagnosis, pathology, differential diagnosis, and treatment are described. The treatment of simple trachroma is often confined to expression of any large follicles and the use of mild astringents. In infected trachroma the conjunctivitis should be treated with antibiotic and chemotherapeutic agents. (17 references) G. S. Tyner.

Coverdale, H. **Pterygium.** Tr. Ophth. Soc. New Zealand (Supp. N. Z. M. J.) pp. 5-7, 1953.

The incidence of pterygium varies geographically, but no correlation can be made with specific geographic factors. Pterygium is as common in New Zealand as senile cataract. The only nearly constant concomitant observed was mild chronic conjunctivitis. Reaction from operation is much reduced if the conjunctivitis is first controlled. Recurrences are far less common since a McReynolds operation, modified to leave 3 or 4 mm. of bare sclera, has been used. Robert A. Moses.

Esente, I. **Lamellar keratoplasy.** Gior. ital. oftal. **7**:119-140, March-April, 1954.

Twenty-four cases treated with lamellar keratoplasty are described in detail. These included various conditions of corneal opacity, and one recurrent pterygium. Results were generally favorable but there was one case of corneal abscess behind the graft, and another of traumatic cataract due to perforation of the cornea during operation. Best results were obtained when there was little or no vascularization. In these cases, the author advises irradiation with beta rays, both before and after operation. Astigmatism after operation should be treated with contact lenses.

The advantages of lamellar keratoplasty over simple keratectomy or performing keratoplasty are discussed and its values for preparing for perforating corneal grafting is stressed. (22 figures, 29 references) V. Tabone.

Kok-van Alphen, C. C. **Cornea.** Transplantation Bull. **1**:136-137, July, 1954.

Homografts of sclera, skin and mucous membrane were transplanted into the rabbit's cornea. The grafted eyes remained quiet for about two weeks, then exhibited an ophthalmitis for about one month. The transplants then began to clear, and after 9 to 14 months the transplanted tissue appeared to be changed grossly into an opaque corneal tissue. Microscopically,

cornea-like stroma, corneal epithelium, Descemet's membrane, and some endothelium were found. Irwin E. Gaynor.

Malamud, B. **Epidemic keratoconjunctivitis.** Arch. oftal. Buenos Aires **29**:114-131, Feb., 1954.

This is an excellent review which covers in full detail the whole subject of epidemic keratoconjunctivitis. (2 figures, 34 references) A. Urrets-Zavalía, Jr.

North, D. P. **The treatment of interstitial keratitis.** Brit. M. J. **2**:7-9, July 3, 1954.

Among 20 cases of interstitial keratitis as a manifestation of congenital syphilis in which cortisone and adequate antisyphilitic measures were used, eight occurred in children who had no previous keratitis and no impairment of vision before the attack for which they were under treatment. These eight cases are described in detail. The use of cortisone has changed syphilitic interstitial keratitis from a long and painful illness, with the sequelae of defective vision or blindness, into an actute condition lasting only a few days. The condition is one which can easily be kept under control until the eyes are quiet and the vision is restored. Except for very acute cases, only out-patient attendances are required. If treatment can be started before corneal infiltration obscures vision, sight need not be impaired at any time. F. H. Haessler.

Oguchi, M. **Allergic reaction to pollen and vernal conjunctivitis.** Acta Soc. Ophth. Japan **58**:735-739, Aug., 1954.

The pollen was collected from various kinds of plants. It was rubbed into a scarified skin surface of normal man. When a positive skin reaction was observed after 20 minutes, the respective pollen was introduced into the conjunctival sac. A conjunctivitis which looked like a vernal conjunctivitis was brought about in three of the eight cases examined. There was an increase in esosinophiles in the discharge. The pollen of Salix gracilistyla and of Alopeculus aequalis was the agent in these cases and in six cases of latent vernal conjunctivitis in the winter season. The pollen of these two plants was put into the conjunctival sac. A positive reaction occurred in three of the six patients tested. The author concludes that vernal conjunctivitis may be an allergic reaction to the pollen of various plants. (2 tables, 9 references) Yukihiko Mitsui.

Pittar, C. A. **A new method of corneal grafting.** Tr. Ophth. Soc. New Zealand (Supp. N.Z.M.J.) pp. 22-28, 1953.

Donor cornea is excised, placed epithelium down, on a paraffin wax block and a disc punched out by pressing a trephine through it (after Amsler and Verrey). A trephine guide, similar to the splint to be used later, is sutured to the recipient cornea, and a trephine mark made on the cornea. The guide is removed, leaving the sutures. The mark is stained. A Saemisch type section along a diameter of the incised circle, and just longer than the diameter, allows the edge of the punch blade to be inserted under the cornea. The precision-made punch consists of a trephine and a disc-shaped blade attached to a rod. Closing the handle of the instrument draws the blade into the lumen of the trephine. Enough cornea is punched out above and below the section to admit the entire blade of the punch, and the punch is again closed. The graft is placed and held with a circular splint, utilizing the sutures previously laid for the trephine guide. Robert A. Moses.

Rossi, Antonio, **Keratoglobus and its relation to keratoconus and megalo-cornea.** Rassegna ital. d'ottal. **23**:220-231, May-June, 1954.

Rossi points out the numerous confusing, and often contradictory, terms

applied to malformations affecting the cornea and the anterior portion of the sclera. He describes the case of a 24-year-old man, studied for several years, in whom it was necessary to distinguish between keratoconus and keratoglobus in one eye and keratoglobus and hydrophthalmos in the other. The literature is reviewed. (5 figures, 16 references)

Eugene M. Blake.

Valentin Gamazo, Ignacio. **Treatment of infected ulcers of the cornea.** Arch. Soc. oftal. hispano-am. **14**:74-119, Jan., 1954.

This is a comprehensive monograph on the subject, with a complete review of the literature and an extensive bibliography but no new material. (82 references)

Ray K. Daily.

Vannini, Angelo. **Cyst formation in pterygium.** Rassegna ital. d'ottal. **23**:211-219, May-June, 1954.

The cyst walls showed stratified epithelium with cells partly keratinized surrounding the cavity in the pterygium. The origin of the cyst is probably the same as that of the pterygium itself. The inflammation of the zone of conjunctiva which preceded the formation of the pterygium may have been the predisposing cause of the obliteration of the connective tissue and cavity formation. Hayline degeneration of connective tissue bundles and epithelial cells with picnotic changes were present. (8 figures, 5 references)

Eugene M. Blake.

Vaughan, Daniel G., Jr. **Xerophthalmia.** A.M.A Arch. Ophth. **51**:789-798, June, 1954.

The history, clinical characteristics, pathology, and treatment of xerophthalmia are reviewed. Six cases are presented. The disease is apt to occur in infants with avitaminosis A, in children and adults on antiallergic diets and in alcoholics and patients with gastrointestinal and liver diseases. Xerophthalmia characteristically develops in four stages: 1. night blindness, 2. xerosis of the conjunctiva, 3. xerosis of the corneal epithelium and 4. keratomalacia. Intramuscular injections of 100,000 units of vitamin A followed by oral and local use of vitamins is probably the treatment of choice. (38 references)

G. S. Tyner.

Wales, H. J. **Multiple myxofibromata of the cornea.** Tr. Ophth. Soc. New Zealand (Supp. N.Z.M.J.) pp. 38-43, 1953.

Four small white spots, entirely in the cornea, were noted at birth, one at the 3- and the 9-o'clock position in each eye. Beginning at the age of one and one-half years these areas have been excised, apparently completely, several times within a year, but they recurred in a few weeks. The lesions are progressive. The diagnosis is based on microscopic appearance. (4 figures)

Robert A. Moses.

Yagihashi, Y. **Allergic conjunctivitis of phlyctenular type.** Acta Soc. Ophth. Japan **58**:740-749, Aug., 1954.

Sixty cases of phlyctenular keratoconjunctivitis and episcleritis were examined by slitlamp and biopsy. At the beginning of the phlyctenule, there is an acutely exudative change with leucocytic infiltration. This is followed by a chronic proliferative inflammation with lymphocytic infiltration and nodule formation. Ulceration takes place when the leucocytes are replaced by lymphocytes.

In slight scleritis there is only a diffuse hyperemia but in severe cases a nodule resembling a phlyctenule can be observed. The author suggests that a tuberculin-type allergic reaction may be taking place.

Yukihiko Mitsui.

## 8

### UVEA, SYMPATHETIC DISEASE, AQUEOUS

Hope-Robertson, W. J. **Malignant melanoma of the iris.** Tr. Ophth. Soc. New Zealand (Supp. N.Z.M.J.) pp. 28-35, 1953.

A malignant melanoma of the iris in an adult is described, and also a benign melanoma of the iris, which had grown in an 11-year old boy. (7 figures)

Robert A. Moses.

## 9

### GLAUCOMA AND OCULAR TENSION

Bietti, G. B. **The diagnostic problem of glaucoma.** Irish J. M. Sc. **342**:237-249, June, 1954.

Relative anoxia in glaucomatous patients will produce an increase in the field defects and also considerable enlargement of the blind spot. Compression of the eyeball to a value just above the retinal diastolic pressure may be performed in the office while testing with the tangent screen for a period of not more than 10 minutes. Audiometric examination in glaucomatous patients during a provocative test will show a 5 to 50 decibel loss in the 4,000 to 10,000 frequency range, which returns to normal as soon as the anoxia or compression is reduced. Smoking one or two cigarettes may make the water-drinking test much more positive and at the same time, tell the doctor whether or not a certain glaucoma patient may smoke. (9 figures, 39 references) Irwin E. Gaynon.

Grant, W. M., and Trotter, R. R. **Diamox (acetazoleamide) in treatment of glaucoma.** A.M.A. Arch. Ophth. **51**:735-739, June, 1954.

Diamox apparently has no serious systemic or ocular side effects when used for a short time. In doses up to 1,000 mg. daily patients may notice paresthesias of the extremities. These symptoms quickly disappear when the drug is stopped. No "tapering-off" period is necessary. When high doses of the drug are used there is apparently a shift in the systemic acid-base balance.

The drug seems most effective as a supplement to local miotics in the treatment of primary acute narrow-angle glaucoma. It is also helpful as a supplement to standard local medications in the treatment of attacks of glaucoma secondary to iridocyclitis. It is least effective in the treatment of chronic simple wide-angle type glaucoma and in secondary glaucoma when the angle is occluded by neovascularization or peripheral anterior synechiae. (2 references) G. S. Tyner.

de Roetth, Andrew, Jr. **Relation of tonography to phasic variations of intraocular pressure.** A.M.A. Arch. Ophth. **51**: 740-749, June, 1954.

It was demonstrated by tonography that eyes with chronic simple, wide-angle type glaucoma do not show an increased facility of aqueous outflow after a spontaneous or induced rise in diurnal pressures. This is evidence to support the belief that mechanical obstruction due to organic angle changes is the cause of this type of glaucoma as opposed to the functional or neurovascular theory. The study emphasizes the practical importance of early morning determinations of tension in the diagnosis of glaucoma. The study also supports the contention that the water-drinking test is based on sudden dilution of the blood. The osmotic pressure of the blood is lowered so that more water impinges on the blood-aqueous barrier and this results in a greater flow of aqueous into the anterior chamber. Since there is a constant rate of flow of aqueous out of the anterior chamber of any given eye, the glaucomatous eye shows a rise in ocular tension. (5 tables, 10 references) G. S. Tyner.

Wilson, R. P. **Capsular exfoliation and glaucoma capsulare.** Tr. Ophth. Soc. New Zealand (Supp. N.Z.M.J.) pp. 8-21, 1953.

The history and clinical aspects of the subject are reviewed. The present material consists of 27 patients (51 eyes). Seven of the two-eyed patients had capsular exfoliation in one eye only. Eight patients,

average age 72 years, had exfoliation without hypertension or other evidence of glaucoma; five whose average age was 73 years, had exfoliation without hypertension but with field defects of a glaucomatous type, and 14 patients, average age 71 years, had exfoliation with hypertension and glaucomatous field defects. The average age of all patients with chronic glaucoma seen at the Dunedin Hospital was 64 years. The incidence of capsular exfoliation in all primary glaucoma was 18 percent and 48 percent of patients seen with capsular exfoliation did not have hypertension. From these figures and other arguments it is concluded that capsular exfoliation is not the essential cause of glaucoma capsulare. Treatment is the same as for ordinary glaucoma simplex.

Robert A. Moses.

## 10

### CRYSTALLINE LENS

Alajmo, A., and Ambrosio, A. **The permeability of the capsule of the lens after the application of diathermy to the sclera.** Gior. ital. oftal. 7:203-211, May-June, 1954.

Experiments on the eyes of rabbits showed that the application of diathermy to the sclera produced only transitory increase in the weight of the lens. There was no clear evidence that the application of diathermy in cases of detachment of the retina was in any way responsible for the few cases of cataract that sometimes occur after this type of surgery. (3 tables, 9 references) V. Tabone.

Laje Weskamp, R., and Piccardi, R. **Posterior lentiglobus.** Arch. oftal. Buenos Aires 29:101-113, Feb., 1954.

Posterior lentiglobus is a generally uniocular, hemispherical projection of the posterior surface of the lens, which nearly always occurs in association with some axial opacities of the posterior cortical layers and is located at or near the posterior pole (only two peripheral cases are to be found in the literature). When the red reflex of the fundus is observed through a dilated pupil, the malformation is seen as a dark disc, whose appearance was compared by Knapp to that of an oil drop. A sharp circular reflex, caused by a reflection from the capsule at the conus margin, is characteristically brought out by slitlamp examination (Vogt). It is unfortunate that no mention is made of that curious phenomenon described by Vogt (Atlas der Spaltlampenmikroscopie. Berlin, Springer, 1931, vol. 2, p. 452) under the name of "Scherenreflexe," an explanation of which has been given recently by Franceschetti and Rickli (Bull. Soc. Franc. d'ophtal. 64:168-175, 1951) by means of a plastic model. The condition must be considered congenital and of a developmental origin; the mechanism of its production is still not fully understood.

A typical case of this rare anomaly is presented as occurring in the right eye of a 24-year-old man, whose other eye exhibited a form of congenital cataract. (4 figures, 21 references)

A. Urrets-Zavalía, Jr.

Montanelli, Mario. **Congenital ectopia of the lens.** Gior. ital. oftal. 7:176-182, March-April, 1954.

Four cases of ectopia of the lens are described in detail. The etiological theories and the influence of circulatory disturbances, heredity, hormonal imbalances, intracranial inflammation and changes in the region of the diencephalon and hypophasis are discussed. Congenital dislocation of the lens, even if found by itself, should be considered as part of the well known syndrome of Marfan. (2 figures, 2 references) V. Tabone.

## 11

### RETINA AND VITREOUS

Bellavia, M. **Experiments on vitreous transplantation.** Gior. ital. oftal. 7:225-234, May-June, 1954.

After an interesting survey of the published attempts at vitreous transplantation, similar experiments on eight dogs are described. One group received fresh vitreous, while a second group received refrigerated vitreous. The first group showed a successful, clear transplant in 33 percent of the cases; in the second group, there were no cases of a totally clear transplanted vitreous and in 25 percent of the cases, there was a dense opacity. The resistance of the vitreous to foreign substances is stressed, but the cases which were successful give hope that further refinement in technique may improve the prospects of vitreous transplantation is suitable cases. (33 references)

V. Tabone.

Bellavia, M., and Pellegrino, F. **Experimental retinitis pigmentosa and vitamin $B_{12}$.** Gior. ital. oftal. 7:167-175, March-April, 1954.

Retinitis pigmentosa was produced experimentally in nine rabbits by the intravenous injection of sodium iodate; when the degeneration of the retina was equally pronounced in all of them, they were divided into three groups. The first received ten daily retrobulbar injections of 10 mg. vitamin $B_{12}$, the second group received the same plus a daily intramuscular injection of 5 mg. vitamin $B_{12}$ for ten days, and the third group received no treatment. Ten days after cessation of treatment all the rabbits were killed and the eyes examined histologically. The eyes of the first two groups showed a less marked retinitis, both in degree and extension. The ways in which sodium iodate can produce retinitis pigmentosa and how vitamin $B_{12}$ can protect the retina are discussed. (11 references) V. Tabone.

Bonaccorsi, A. **Serous central retinitis of Masuda.** Gior. ital. oftal. 7:249-275, May-June, 1954.

After an extensive review of the published cases of this disease, the author describes two cases of serous central retinitis. The symptomatology is given in detail, and stress is laid on the lack of color inversion of consecutive images during the whole course of the disease. This may be a very useful finding in early doubtful cases. The hypothesis is advanced that toxins passing through the retina sensitize both the pigmentary epithelium and the choriocapillaris, with the production first of a serous fluid, and later of a turbid exudate. There was complete resolution in both cases. (4 figures, 44 references) V. Tabone.

Carreras Duran, B. **Periphlebitis of the retina and toxoplasmosis.** Arch. Soc. oftal. hispano-am. 14:49-51, Jan., 1954.

The author reports a case of periphlebitis of the retina in a women, 21 years old, in whom the only positive finding in a thorough general examination was a positive reaction to the Sabin and Feldman test for toxoplasmosis, with a dilution of 1/64. A study of the blood chemistry revealed a hypoalbuminemia, with a diminution of the alpha globulins, and an increase in the gamma globulins. The author suggests that other cases of doubtful etiology may be due to the same cause. The toxoplasmosis infection could produce the disturbance in the protein composition of the plasma and a change in the permeability of the endothelium of the vessel walls with consequent retinal hemorrhages and foci of choroiditis. The author suggests that an investigation for toxoplasmosis and a study of the plasma protein be made in cases of recurrent vitreous hemorrhages and retinal periphlebitis. (1 reference)

Ray K. Daily.

Cowper, Alexander R. **Angioid streaks.** A.M.A. Arch. Ophth. 51:762-782, June 1954.

This is a comprehensive article on the subject which includes two case reports. The author favors the opinion that the ocular findings are part of a degeneration

of a systemic elastic tissue which produces generalized vascular disease. He believes the weight of evidence favors the opinion that the fundus abnormality results from a primary vascular disorder in the choroid which leads to secondary and incidental ruptures in Bruch's membrane, bearing no ophthalmoscopic relationship to the angioid streaks. The streaks probably result from choroidal hemorrhagic extravasations accompanied by pigment migration. (3 figures, 75 references) G. S. Tyner.

Hope-Robertson, W. J. **Radiological examination in retinoblastoma.** Tr. Ophth. Soc. New Zealand (Supp. N.Z.M.J.) pp. 36-38, 1953.

X-ray photographs may reveal calcium deposits in retinoblastomas and are an aid in the diagnosis. An illustrative case is presented. (2 figures)

Robert A. Moses.

Minsky, Henry. **Correlation of ocular changes in essential hypertension with diastolic blood pressure.** A.M.A. Arch. Ophth. **51**:863-874, June, 1954.

The author believes after a 10 year study that the severity of retinal changes in hypertension parallels the diastolic pressure. He presents a system of study and grading of the retinal changes and their correlation with the diastolic pressure. (4 charts, 7 references)

G. S. Tyner.

Rama, Giovanni. **Clinical and therapeutic considerations of detachment of the retina.** Rassegna ital. d'ottal. **23**:189-204, May-June, 1954.

In the first part of the paper the author reviews and discusses the pathogenetic significance of the fundamental clinical data observed in cases of retinal detachment in his clinic during the last three years. In the second part he presents the therapeutic orientation of the school to which he adheres. He expresses the opinion that surgical intervention must be attempted even when the probability of success is slight. The indications, the technical method employed and the factors which permit prognostic judgement and treatment are explained. The technique must not be standardized, but must be adapted to the individual problem. Finally preventive surgery is discussed. (3 figures, 32 references)

Eugene M. Blake.

Rubino, A. **Chemistry of sub-retinal fluid in Coats' disease.** Gior. ital. oftal. **7**:191-198, May-June, 1954.

The author analyzed the subretinal fluid in a case of Coats' disease, with a view of determining its origin. Examinations included the estimation of glucose and nitrogen content, as well as the determination of the pH, electrophoresis and the absorption spectrum of ultraviolet radiation. The reaction of Rivalta was positive, and the findings indicated that the fluid was very much like blood serum. Further work is necessary before a definite opinion on the origin of the fluid could be given. (5 figures, 8 references)

V. Tabone.

Yoshioka, H. **Studies of the eyes of premature infants.** Acta Soc. Ophth. Japan **58**:879-904, Aug., 1954.

Necropsy examination of 14 premature infants showed a rosette formation, edema, hemorrhage and proliferation of the glia tissue and the vessel endothelium in the retina in ten cases. There was a protracted physiological degeneration in the fetal zone of the paranephros. The relationship between the development of retrolental fibroplasia and the function of the paranephros is discussed. (22 figures, 13 tables, 161 references)

Yukihiko Mitsui.

## 13

## NEURO-OPHTHALMOLOGY

Bocci, Giorgio. **Aneurysms of the cavernous sinus and of the internal carotid.**

Gior. ital. oftal. 7:101-118, March-April, 1954.

After an interesting survey of carotid aneurysms, their classification and difficulty in diagnosis, two cases are described, one of arterio-venous aneurysms in the cavernous sinus, and another of aneurysms of the internal carotid below the clinoid process. The former showed no pulsation. There was a slight proptosis of the other eye four months after the accident which caused the original lesion. No surgical treatment was given because compression of the carotid was not well tolerated. To help re-establish circulation, after the blood in the aneurysm has coagulated into a fibrinous mass, heparin was given. The patient recovered.

The second case, was treated surgically, first by incomplete ligature of the common carotid, followed by complete ligation of the internal carotid of the same side. The author plans to completely tie the internal carotid at a future date. (39 references)

V. Tabone.

Kawahata, H. **A study of the pupillary reflex.** Acta Soc. Ophth. Japan **58**:841-850, Aug., 1954.

To record an action current of the pupil (an electropupillogram) is possible only when the electric poles are put on the cornea. The electropupillogram and pupillary contraction are parallel to each other, but there is no development of current by a re-dilation of the pupil. The author discusses the relationship between the change in the curve of the electropupillogram and the change in the light stimulus which causes the light reflex. (13 figures, 5 tables, 15 references) Yukihiko Mitsui.

## 14

## EYEBALL, ORBIT, SINUSES

Adrogue, E., and Garcia Nocito, P. F. **Hydatic cysts in the orbit.** Rev. oto-neuro-oftal. Sudam. **29**:37-42, March-April, 1954.

The authors give a complete summary of the parasitology of tenia ecchynococcus. Eighty percent of all human infestations occur in patients below 40 years of age. Pain is usually the first sign, followed by diplopia and exophthalmos. The exophthalmos is usually progressive in type and is always unilateral. The cysts do not always have the same localization in the orbit, but have a tendency to insinuate themselves between the globe and the bony orbital wall. Transillumination many times is very helpful in the diagnosis. The only effective treatment is the surgical removal of the cysts preceded by treatment of the cysts by formol, in order to kill the escolex and to prevent serious anaphylactic reactions. The only biological test which is somewhat reliable is the intracutaneous test of Cassoni.

The complications of the untreated cyst are of two types: mechanical and toxic. The mechanical complications consist of keratitis due to lagophthalmos. The toxic manifestations may be spontaneous, due to fissures in the cysts or may follow surgical punctures, with very severe allergic manifestation and severe shock. If the cyst is not excised, the prognosis is grave; eventually there will be complete blindness. Walter Mayer.

Fernandez, A. G., Ortiz de Landazuri, E., and Carreras Matas, B. **Exophthalmos caused by thiouracil.** Arch. Soc. oftal. hispano-am. **14**:52-57, Jan., 1954.

The authors report a case of exophthalmic goiter, in a young man of 24 years, who after nine days of therapy with thiouracil developed a marked increase in exophthalmos, while the other symptoms were improved. X-ray treatment of the pituitary region showed an enlargement of the sella turcica. The improvement in the general symptoms and the aggravation of the exophthalmos indicate that some factor other than the thyroid was responsible for the exophthalmos. The pituitary factor probably accounted for the exoph-

thalmos, and the thiouracil which suppressed the inhibitory action of the thyroid on the pituitary permitted the hyperactivity of the pituitary to attain greater proportions. (4 figures, 6 references) Ray K. Daily.

Galvez Montes, J. **Primary myeloma of the orbit.** Arch. Soc. oftal. hispano-am. **14**:58-64, Jan., 1954.

This is a report of the case of a 41-year-old man who had a primary orbital myeloma, which in spite of an orbital exenteration was fatal within ten months because of thoracic metastasis. There were no general symptoms or laboratory signs to suggest the diagnosis, and it is emphasized that a biopsy for diagnosis should be made early, because the earlier treatment is instituted the more effective it is apt to be. (2 figures, 4 references) Ray K. Daily.

King, John H. **A new buried movable orbital implant.** U.S.A.F. Med. J. **5**:835-838, July, 1954.

The author used a modification of the Lee Allen implant in 67 cases with good result. The anterior surface of the implant is covered with a stainless steel mesh. Sutures are tied separately over the mesh of the implant. (2 figures, 1 reference) Irwin E. Gaynon.

Matteucci, Pellegrino. **Biomicroscopic changes common to several ocular manifestations.** Rassegna ital. d'ottal. **23**:205-210, May-June, 1954.

The biomicroscope reveals teratological changes in the mesodermal tissue in the angle of the anterior chamber in microphthalmos, microcornea, aniridia, and coloboma of the iris. Anomalies of the episcleral and ciliary circulation are also present. No aqueous veins were observed in these cases, but in the region of the limbus there were irregular, tortuous vessels, containing blood. Neovascularization was lacking.

Rupture of Descemet's membrane was observed in eyes with microcornea and coloboma of the iris. In microphthalmos and coloboma the angle was wide, the trabeculum was visible and almost always there was undifferentiated tissue. (5 figures, 3 references) Eugene M. Blake.

Naquin, Howard A. **Exenteration of the orbit.** A.M.A. Arch. Ophth. **51**:850-862, June, 1954.

The cases of exenteration of the orbit (48) performed at the Wilmer Institute during the past 25 years are reviewed. Surgical technique, operative complications and final outcome are discussed. In cases of orbital extension of choroidal melanoma only two of nine patients were alive after three years. It is probably unnecessary to remove the lids in these cases; neither is it necessary in meningiomas. In orbital sarcomas the lids should be sacrificed. The procedure of exenteration was more easily accomplished if there had been a prior enucleation. Kirby's technique of an immediate split-thickness skin graft onto the bare bone of the orbit seems well advised. (4 figures, 11 tables, 17 references) G. S. Tyner.

Vannini, A., and Pettinate, S. **Radiography of the orbital veins.** Rassegna ital. d'ottal. **23**:241-246, May-June, 1954.

The radiologic visualization of the venous orbital circle and the cavernous sinus, which is made possible by the injection of a radiopaque contrast medium by way of the angular veins of the face, furnishes interesting data in diverse affections of the orbital region. The medium used is called m.d.c. and 8 to 10 cc. of the 35 percent solution is injected rapidly and the radiogram is made during the course of the injection. A lateral projection gives better results than a frontal picture. The method is especially valuable in the study of orbital tumors. (3 figures, 4 references) Eugene M. Blake.

# NEWS ITEMS

Edited by Donald J. Lyle, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

## Deaths

Dr. Henry Minsky, New York, New York, aged 59 years.

Dr. Bascom Headen Palmer, Miami, Florida, died September 2, 1954, aged 64 years.

Dr. Merritt Whitacre Wheeler, Stillwater, Minnesota, died June 21, 1954, aged 67 years.

## Announcements

### Call for papers

The 1955 joint meeting of the Section on Ophthalmology of the American Medical Association and the Association for Research in Ophthalmology will be held in Atlantic City, New Jersey, June 6 through 10, 1955.

Anyone wishing to present a paper before the Section on Ophthalmology is urged to communicate with Dr. Harold G. Scheie, secretary, 313 South 17th Street, Philadelphia 3, Pennsylvania, as soon as possible. A title and abstract, of not less than 30 nor more than 150 words, must be in his hands by January 1, 1955.

Anyone wishing to present a paper before the Association for Research should contact Dr. Lorand V. Johnson, secretary, 10515 Carnegie Avenue, Cleveland 6, Ohio, at an early date.

### Contributions to the Knapp Fund

Members of the Section on Ophthalmology of the American Medical Association are reminded that their contributions to the Knapp Fund are now due and payable to Dr. Parker Heath, 243 Charles Street, Boston 14, Massachusetts. A contribution of $10.00 entitles the contributor to a volume of the 1954 *Transactions*. Contributions in excess of $10.00 are invited.

### Bowman Lecture

Dr. John H. Dunnington, New York, New York, has been invited to give the Bowman Lecture at the annual congress of the Ophthalmological Society of the United Kingdom which will be held at the Royal Society of Medicine, 1 Wimpole Street, London, W. 1. on April 28, 29, and 30, 1955. Only two other American ophthalmologists have been so honored, Dr. George de Schweinitz and Dr. Arnold Knapp.

### Research Study Club

From Monday to Friday, January 24 through January 28, 1955, the eye program of the 24th annual midwinter clinical convention of the Research Study Club of Los Angeles will be presented in Los Angeles. The preceding week, January 17 through January 22, 1955, will be devoted to the ear, nose, and throat program. Each applicant for attendance at the convention must be a member in good standing of the American Medical Association in order to become eligible for attendance. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. Make your check payable to the Midwinter Clinical Convention and mail to: Dr. Pierre Violé, treasurer, 1930 Wilshire Boulevard, Los Angeles 57. If anything prevents attendance, this fee will be returned.

Those who do not have plans for a place to stay are advised to write for reservations to: Mr. H. M. Nickerson, manager of the Elks Club, 607 South Parkview Street, Los Angeles 57.

### Florida midwinter seminar

The ninth annual University of Florida Midwinter seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach the week of January 17, 1955. The lectures on ophthalmology will be presented on January 17th, 18th, and 19th and those on otolaryngology on January 20th, 21st, and 22nd. A midweek feature will be the midwinter convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 19th, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at eight p.m. on Wednesday. The seminar schedule permits ample time for recreation.

The seminar lecturers on ophthalmology this year are: Dr. William F. Hughes, Jr., Chicago; Dr. Phillips Thygeson, San Jose; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis; and Dr. Milton L. Berliner, New York. Those lecturing on otolaryngology are: Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler, Jr., New York; Dr. Arthur W. Proetz, Saint Louis; and Dr. David D. DeWeese, Portland, Oregon.

### New Eyes for the Needy

New Eyes for the Needy, Short Hills, New Jersey, is a nonprofit, volunteer charity that has helped more than 85,000 people to better sight. Its work is done entirely through the collection of old glasses and jewelry; funds are never solicited.

The American Academy of Ophthalmology and Otolaryngology is sponsoring New Eyes for the Needy at next year's convention. The group also

has the approval of the National Society for the Prevention of Blindness.

### Japan Medical Congress

The XIV Japan Medical Congress will convene at Kyoto, Japan, April 1 to 5, 1955. The Japan Travel Bureau, c/o Kyoto Station Hotel, Kyoto-ekimae, Shimokyo-ku, Kyoto, will arrange for travel to Japan, tour within the country, and everything concerned with travel, including hotel accommodations, for those who plan to attend the congress.

## Societies

### Kansas City program

The ophthalmology programs for the 1954-1955 sessions of the Kansas City Society of Ophthalmology and Otolaryngology will be held in the afternoon on the following dates:

November 18, 1954: "Plastic surgery of the lids," Dr. Wendell L. Hughes, Hempstead, Long Island, New York.

December 16, 1954: Dr. Charles L. Schepens, Boston, will present the Curran Lecture.

January 20, 1955: To be announced.

February 17, 1955: "Strabismus," Dr. Marshall M. Parks, Washington, D.C.

March, 1955: Out-of-town trip, date and place to be announced.

April 4 to 8, 1955: Postgraduate course, University of Kansas Medical Center. Guest speakers: Dr. James H. Allen, New Orleans; Dr. A. R. Irvine, Los Angeles; and Dr. V. Everett Kinsey, Detroit.

May, 1955: Social meeting, date and place to be announced.

Note: Ophthalmology and otolaryngology meetings will be held at General Hospital No. 1, Kansas City, Missouri, on November 18, 1954, and January 20, 1955. The remainder of the meetings will be held at the University of Kansas Medical Center.

Officers of the society are: President, Dr. William B. Barry; president elect, Dr. Donald O. Howard; vice-president, Dr. Barnard C. Trowbridge; secretary, Dr. Dick Underwood; treasurer, Dr. Fred Bosilevac.

### East Central Research Program

The East Central Section of the Association for Research in Ophthalmology will meet on Monday, January 10, 1955, at the University of Buffalo, Medical School, Buffalo, New York. Registration will be at 8:30 a.m. and on the program starting at 9:00 a.m. will be the following papers:

"The presence of a nonspecific factor in experimental uveitis," Ted Suie and Frank W. Taylor, Department of Ophthalmology, The Ohio State University; "Some new evaluations of lower-case letters in relation to reading," J. H. Prince, Department of Ophthalmology, Ohio State University; "Comparative tonographic study of normotensive eyes of whites and Negroes," B. Boles-Carenini, R. E. Buten, W. M. Spurgeon, and K. W. Ascher, University of Cincinnati, College of Medicine; "Comparative tonographic studies on right and left normotensive eyes," B. Boles-Carenini, R. E. Buten, W. M. Spurgeon, and K. W. Ascher, University of Cincinnati, College of Medicine; "A comparative evaluation of some newer topical anesthetic agents," E. Kenneth Vey and Jay G. Linn, Jr., Pittsburgh.

"Effect of fatigue and stimulants on binocular fusion," Gerhard A. Brecher, Edward Purnell, and William Hoover, Western Reserve University, School of Medicine, Cleveland; "Effect of sympathetic nerves on the ciliary muscle," Carlton H. Melton and Gerhard A. Brecher, Western Reserve University, School of Medicine, Cleveland; "The effect of protein and protein derivatives on the development of galactose cataracts," John W. Patterson, Western Reserve University, School of Medicine, Cleveland; "The intensity discrimination of the frog retina," Leo E. Lipetz, Department of Ophthalmology, Ohio State University.

"A lens for the encouragement of simultaneous macular perception," K. Elizabeth Pierce Olmstead, Buffalo; "Fundus changes after cataract surgery," A. Dellaporta, University of Buffalo Medical School; "Experimental studies on artificial detachment of the choroid," A. Dellaporta, University of Buffalo, Medical School; "The effect of antiglare devices on color discrimination," Clement McCulloch, R. E. White, and N. C. Turnour, Toronto; "An experimental study of electroretinography: II. A study of pure population retinas," Julius Praglin and Albert M. Potts, University Hospitals of Cleveland; "The penetration of cortisone and hydrocortisone into the ocular structures," S. Hamashige and Albert M. Potts, University Hospitals of Cleveland; "The transfer of corneal water as measured with the aid of tritium oxide," Albert M. Potts, Doris Goodman, and L. V. Johnson, University Hospitals of Cleveland; "Are subjective variations in visibility significant?", Sylvester K. Guth, General Electric Company, Nela Park, Cleveland.

"Electro-encephalography with photic stimulation in strabismus," J. C. Ball, University of Toronto; "Application of new tissue culture techniques to the study of herpetic infections of the eye," Frances Doane, University of Toronto; "Growth of conjunctiva in tissue culture," Ann Fowle and Anne Cockeram, University of Toronto; "Influence of immunity on the histology of herpetic keratitis in rabbits," R. L. Hall, University of Toronto; "Studies with Diamox in experimental cyclitis in rabbits," K. E. Schirmer, University of Toronto.

"Visual function studies in a case of large aberrant macular vessels," David Volk, Western Reserve University, Cleveland; "Corneal wound repair as followed by silver carbonate stain technique," Irving Shapiro, University of Michigan; "Syndrome of Pachydermoperiostitis, general and ocular manifestations," Dr. Robert O. Reisig; "A second family of keratosis spinulosa cum ophiasi," Dr. Frank B. Galyon; "Waardenberg's syndrome," Dr. Harold F. Falls, University of Michigan.

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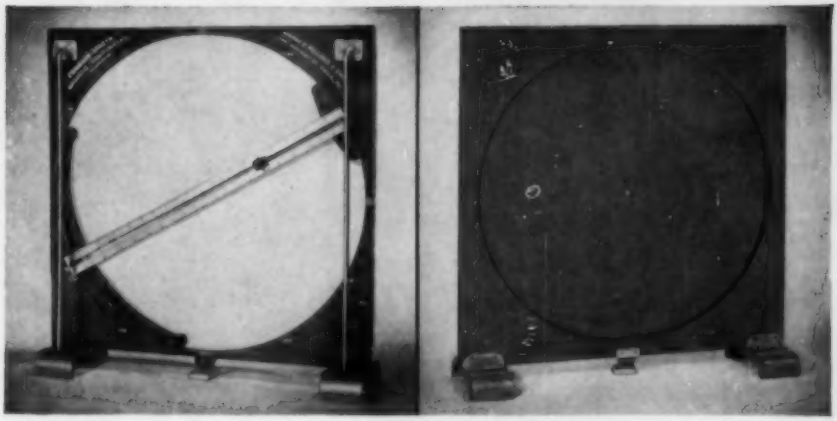

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